BRAIN.

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## Original Articles and Clinical Cases.

# ANEURISMS OF THE LARGER CEREBRAL ARTERIES.

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My subject is "Aneurisms of the Larger Cerebral Arteries," with special reference to their possible diagnosis and treatment. This resolves itself into the question of symptomatology of intracranial aneurisms. Is it possible, from the symptoms produced, to form a diagnosis of aneurism, and of its situation when occurring within the cranial cavity?

By aneurism, I mean *true* aneurism of the arteries of the brain. I do not include arterio-venous aneurism of the internal carotid artery in the cavernous sinus, a subject that has been ably discussed by Walter Rivington (50) and Adams Frost (17) in this country, and by several writers on the Continent. Further, what remarks I have to make will be made almost solely from cases of aneurism whose existence has been proved by a *post-mortem* examination.

My comments will be based on the notes of 555 cases of aneurism of the cerebral arteries found after death. It may be well, perhaps, if I state at the outset the source of this

material. Of this large number, 441 have been recorded in various medical journals and periodicals; 209 were published in Great Britain, forty-four in Canada and the United States of America, and 188 on the Continent,[1] mostly in France and Germany. The remaining 114 cases may be regarded as fresh material. This is made up of seventy-four cases from museums in London and elsewhere, the only publicity of which is the description in the catalogues of the museums; thirty occurred in Colney Hatch Asylum, and notes of the remaining ten were received from friends.

Of these cases of intracranial aneurism, I have seen the actual specimens in 140 instances; 113 are contained in the Pathological museum of this College and in the museums of the eleven leading medical schools in London, in connexion with which I wish to thank the committees of the several hospitals for permission to examine the specimens, and to express my obligations to the curators who rendered me assistance. Of the remainder, over twenty occurred at Colney Hatch Asylum during the fourteen years I was a medical officer in that institution, and the others were sent to me.

I do not propose to enter into detail as to what symptoms may or may not be caused by aneurisms of the cerebral arteries. For all practical purposes these aneurisms can be classed into four groups. (1) Those in which the first indication of a cerebral lesion has been an apoplectic attack due to rupture of the aneurismal sac. (2) Those in which a fatal apoplexy has been preceded by symptoms suggesting a cerebral tumour or other cerebral lesion. (3) Those in which there have been indications of a cerebral tumour only. (4) Those that gave rise to no symptoms whatever during life, and the aneurism was discovered accidentally after death.

There is no distinct relation between the size of an aneurism and the group into which the case falls, though it naturally follows that the larger the aneurismal tumour, the more likely is it to give rise to pressure symptoms during its growth. But there are many cases in which an aneurism of

[1] In looking up the foreign records I have received much help from Mr. T. W. P. Lawrence.

considerable size has been found after death that did not give the slightest indication of its presence during life. It may be regarded as a maxim that the production of symptoms depends more upon the situation of an aneurism than upon its mere size; but here, again, aneurisms even of large size, have been found in regions where one might imagine that signs of nerve compression would surely be manifested, and yet no such signs were present.

Usually an aneurism of the cerebral arteries ruptures while its walls are delicate, and before it has attained a size larger than a marble. Supposing such an aneurism lay against the root of a cranial nerve, its soft and flexible wall need not necessarily cause compression on the adjacent cerebral tissue or nerve root; for what pressure there is would obviously be exercised in the direction of least resistance, that is, into the loose tissues of the arachnoid membrane that abounds in those places where aneurisms are most common.

The aneurisms which give rise to cerebral and nerve symptoms are mostly the slow-growing, thick-walled sacs, which often attain considerable dimensions. There is evidence to show that these aneurisms go on for many years, and that more often than not they do not rupture, death in such cases being brought about either through injury inflicted on the cerebral tissue—softening, disintegration, &c.—or from some intercurrent malady. Occurring in certain places, such an aneurismal tumour may cause sudden death merely through pressure on some vital spot, or death, more or less sudden, may follow apoplexy from the rupture of an artery elsewhere in the brain.

Analysing the 555 cases, I find that in 257, or 46·3 per cent., the first symptoms were those of apoplexy. In many of these cases the patient had, to all appearances, been in good health up to the moment of the attack. In 115, or 20·7 per cent., signs suggestive of a tumour, or which in the light of subsequent events could be attributed to it, had existed previously to a fatal apoplectic stroke. In some of these the signs were very trivial, and might certainly have been produced by other lesions than tumour. In very few indeed

was a tumour actually diagnosed. In 91, or 16·39 per cent., there was no cerebral or meningeal hæmorrhage, but signs and symptoms of a brain tumour or lesion of some kind had been present. In most of the remaining ninety-two cases no mention is made of any clinical condition referable to the aneurism, though in a few attention is specially called to the fact that no symptoms were present during life. Among the last there are doubtless many cases which should be classed in one or other previously mentioned group; while among those in which a final apoplexy is alone recorded there are probably some that presented previous cerebral symptoms, but the earlier history is defective. In 78, or 14·05 per cent., the history of the case points to compression of one or more cranial nerves.

Taking the two classes together, those in which signs of a tumour alone existed and those in which such signs were followed by those of apoplexy, we have a total of 206 cases, or 37·11 per cent., in which the aneurism during life made itself apparent by symptoms. This at first sight does not seem so low a proportion as one is wont to believe. But I would point out again that in many of these cases the symptoms were trivial and might readily have been produced by other means than a cerebral tumour, and not even necessarily by a gross lesion in the cranial cavity. For instance, merely headache and giddiness, though in these cases dependent probably on the intracranial tumour, may owe their origin to many other affections, organic or functional.

I would also point out that in a very considerable number of cases where symptoms of various kinds occurred other lesions have been found in the brain besides an aneurism; for instance, old hæmorrhages or softenings quite unconnected with the aneurismal tumour, though due like that to disease of the vascular system; in some syphilitic subjects there have been gummatous deposits in the brain, or on other vessels than the one that was dilated. Those who have recorded cases have rarely gone to the trouble of differentiating between the symptoms that might have resulted solely from the vascular dilatation. In some cases this could be done with a fair amount of ease; in others, which form

the majority, the task would be extremely difficult, or even impossible.

In 372 instances, or in 67 per cent. of all cases, death was preceded by an apoplectic seizure. In 339 of these cases it was due to rupture of the aneurismal sac. The difference between the two figures (thirty-three) is made up of cases in which fatal hæmorrhage occurred from some other artery of the brain independent of the aneurism.

Among the cases where rupture of the sac with the consequent apoplexy was the first symptom by which the aneurism made its presence known, some have attained a considerable size before this occurred. In the University College Museum is a specimen of aneurism from the middle cerebral artery (No. 3475D) where the sac measures 2 cm. in diameter. It is from a woman aged about 55, who suddenly fell down in the street in an apoplectic attack and died five hours later. In the very similar specimen which I received from my friend Dr. Charles Heaton, of Westgate (Royal College of Surgeons, No. 3795G); here the only previous symptom was an occasional slight headache. The patient, a woman aged about 50, went to lie down on account of headache, and an hour or two later she was found lying on her bed in an unconscious state and died before medical assistance arrived. The aneurism, which measures 2·5 cm. across, was situated on the left Sylvian artery; it had ruptured into the ventricle of the cerebral hemisphere, and no blood had escaped to the base of the brain. The heart, kidneys, and cerebral arteries all appeared healthy.

Many other cases might be cited of such aneurisms without premonitory symptoms. There seems a special tendency for cerebral aneurisms of embolic origin to rupture at an early stage, and this is not surprising, seeing the weakened state of the vessel wall that must result from the inflammatory action set up. There has sometimes been a slight strain or exertion immediately preceding a rupture, but not always. Cases have been recorded where the patient has been resting in a chair, or, as in the case just mentioned, lying on a bed, when the fatal rupture took place.

As an instance of the difficulty in deciding which symptoms

are due to aneurism, and which to other co-existing lesions in the brain, I may point to the specimen of small spherical aneurism at the origin of the inferior cerebellar from the left vertebral, which is now in this Museum (No. 3795I). The little sac, the size of a pea, with hard atheromatous and calcified walls, lies against the roots of the cranial nerves in the ponto-cerebellar triangle. It appears to compress the roots of the left seventh, eighth, and twelfth nerves, and possibly the ninth. The pyramid and olivary body on the same side are markedly atrophied. The basilar is very tortuous and diseased.

The patient, a woman aged 65, was admitted to asylum with organic dementia two and a half years before death. Her intellectual faculties were much impaired. There was some paralysis of the left side of the face, and she was somewhat deaf. She complained of losing power in the hands at times. Weakness in the right arm came on and gradually increased. There was also weakness in the legs, and she dragged the right foot. She had twitching in her limbs, which was most marked when she began to walk. The facial paralysis increased. The lids of the left eye were more or less continually on the twitch, and her eye was always watery. There was indistinctness and stammering in speech, which was not that of general paralysis, but seemed to be due to paralysis in the throat. Saliva collected in her mouth. She now had to be led from place to place, as she was unable to walk by herself. Both as regards her speech and paralysis of limbs she seemed worse on some days than on others. She died a few days after a sudden apoplectic stroke.

A large recent hæmorrhage was found in the lower part of the left cerebral hemisphere, which destroyed the hinder and outer part of the optic thalamus and had broken into the ventricle of the left side. In this same hemisphere was an old hæmorrhage, the size of a hazel nut, beneath the cortex at the outer part of the occipital lobe, and an old patch of softening, the size of a Brazil nut, in the lower part of the temporal lobe. In the right hemisphere was a similar sized patch of softening in the posterior border of

he parietal lobe, and a streak of softening also between the corpus striatum and the claustrum. In the hinder part of the dentate nucleus of the left lobe of the cerebellum was a patch of discoloration from a hæmorrhage of old date.

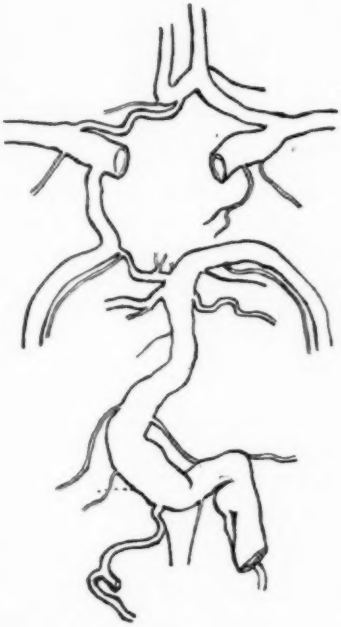


FIG. 1.

Diagram of the larger arteries from the base of the brain, showing an anomalous circle of Willis. The basilar artery is long and tortuous, and mainly a continuation of the left vertebral. On the latter artery, at the origin of the inferior cerebellar branch, is a small spherical aneurism that lay over the roots of the cranial nerves in the ponto-cerebellar triangle. Natural size. Royal College of Surgeons Museum, 3795I.

This case did not come in the series investigated for anatomical variations of the cerebral arteries, but I might point out that the right vertebral—the opposite to that on which the aneurism is situated—is exceedingly small, the basilar being a direct continuation of the left vertebral. At

its upper end the basilar is mainly continued into the left posterior cerebral artery, and the vessel from the carotid that represented the posterior communicating failed to unite with the posterior cerebral. On the right side the posterior cerebral is given off by the internal carotid, and a small communicating vessel connects this with the basilar. The left anterior cerebral is large, and, through the anterior communicating, forms the main origin of the right anterior cerebral (fig. 1).

There is little doubt that the rigid mass formed by the aneurism was the cause of certain of the symptoms, owing to its pressure upon the nerve roots. But, in the presence of the many foci of old hæmorrhages and softenings in the brain, it is impossible to say to what extent it acted in producing atrophy of the pyramid and causing paralysis in the limbs.

A small obsolescent aneurism on the right internal carotid, in which the sac has been converted into a solid calcareous mass about 8 mm. across, is seen in another specimen I have given to the museum (No. 3795E). It was found in a woman with senile dementia. Although the right optic nerve that passed over the tumour is distinctly smaller than that of the opposite side, no appreciable difference was observed in the sight of the two eyes during life. So it has often been when much larger aneurisms have caused considerable destruction of one of the optic nerves. Several of my cases have shown small aneurisms in this situation—that is, immediately the internal carotid has passed through the dura from the cavernous sinus—and some of these have had calcareous walls. In none were there any symptoms which could be attributed to the aneurism. Aneurisms like this have certainly existed a great many years, yet the degenerative changes may possibly be recent compared with the origin of the aneurism.

In St. Bartholomew's Hospital Museum is a specimen (No. 1530B) of aneurism of the left internal carotid in the same situation that is converted into a rigid calcified cyst at least twice the size of that just mentioned. The left optic nerve was flattened and compressed against the inner wall.

The case was that of a woman aged 84, who died of fracture of the skull and laceration of the brain. Nothing was known of any visual affection during life. To me the case seems one of much interest, for had the patient received a somewhat less severe injury, and lived a few years, there is little doubt that the aneurism would have been attributed to the accident.

One might, perhaps, be inclined to think that slowly progressive signs indicative or suggestive of cerebral tumour, with subsequent apoplectic seizure, should make a diagnosis of cerebral aneurism practically certain. But I would warn you against any such rash conclusion. There are plenty of cases on record with such a history, which after death have been shown not to be aneurisms; for example, some cases of malignant growth with hæmorrhage into the tumour or into the meninges. Other cases have occurred of neoplasms or other brain lesions in which death was preceded by symptoms indistinguishable from those of intracranial hæmorrhage, but no blood extravasation was found on examination. Such attacks may even occur in the course of the illness in cases not only of cerebral tumour but of chronic brain disease. I have seen more than one of these cases myself. And I may remind you that in the chronic insane apoplectiform attacks are sometimes observed resembling in detail those due to hæmorrhage, and yet no hæmorrhage has been present. Cases of this kind have sometimes been designated serous apoplexy.

On the other hand, in a certain proportion of the cases of cerebral aneurism, apoplexy has preceded death, but the hæmorrhage has been found to arise from rupture of an artery independent of the aneurismal sac. This was so in at least thirty-three of the cases I have collected. Sometimes the hæmorrhage has occurred inside the brain from a small central artery, but almost as frequently it has been meningeal from a larger vessel in or near the circle of Willis. A good instance of this kind occurred in my own experience.

The patient from whom the large aneurism (Royal College of Surgeons, No. 3795D) was taken was an inmate of Colney

Hatch Asylum on and off for some six years. When first admitted to the asylum in May, 1896, aged 42, she was said to have been going wrong in her mind for eighteen months, but had lately become dangerous to those about her. At that time she had delusions of electrical annoyance, auditory hallucinations and optical illusions. "Electricity was put on her which lifted her into the air. People spoke to her through an invisible tube. She saw imaginary people whom she ran after, trying to stab them with a knife." Her general health was moderate; there was no definite physical defect. At first depressed and melancholic, she brightened up to some extent, and although still deluded, was taken home by her husband in the following December. Two months later she was re-admitted to the asylum, having for a month past been greatly annoyed by "voices." On the certificate it was stated, as a delusion, "She feels a hissing noise in her head which she thinks due to electricity." She was again very depressed, but without suicidal tendencies. There had been no epileptic or other seizure. Her health was fair. There was an extremely slight paresis of the right side of the face; there was also the very slightest tendency for the right eyebal to be diverted outwards and to a still less degree upwards; the palpebral fissure was rather less at the outer canthus on the right side than on the left. All these conditions were so slight that looking at the patient at this time they might easily have passed unnoticed. The patient, again becoming more cheerful, left the asylum in the care of her husband.

In April, 1898, she returned for the third time, after remaining at home five and a half months, during the last two of which mental symptoms had been more pronounced. Electrical persecutions were prominent. The certificate said, "She tells me she is under spiritualism; when she sneezes under the bedclothes she sees bronze flash from her eyes; she says she is being bronzed and electricity applied to her." There was a further development of the right facial paralysis. The ocular fissure on the right side was less open; the right eyeball could not be moved fully outwards.

The patient died in August, 1902, without again leaving

the asylum. During the last two years of life the facial paralysis became more obvious and the right eyeball was turned outwards and upwards in a striking manner. She never appeared to have any defect of vision, but she had marked optical delusions; she saw spirits flying about, at which she would throw anything at hand. She had a persistent delusion that men were under the floor. She was irritable and quick-tempered, and had impulsive outbursts, when she would smash objects, throw chairs about and stamp violently on the floor "to try and kill the men." She sometimes complained that her bread and butter were poisoned. There was no paralysis of limbs. She never had a giddy attack or epileptiform seizure. Speech was unaffected. There was no headache. During the last few months of life she had occasional vomiting, unassociated with food, which, taken with the other signs, was attributed to a cerebral origin. She got thinner and looked ill, but she took her food well, and always said she felt all right. She was put in an infirmary ward about a week only before death. Five days before that occurred there was a sudden apoplectic seizure and she lay subsequently comatose.

An aneurism, forming a tumour the size of a small hen's egg ($1\frac{3}{4}$ ins. long, and $1\frac{1}{8}$ ins. across), was found at the base of the brain (plate i., A, and fig. 2). The aneurism had its connexion with the left internal carotid immediately after its entrance into the cranial cavity. It was mainly situated to the left of the mid-line, but stretched a little over to the right, causing considerable injury to those parts of the brain with which it came into contact. The tumour overlapped the optic chiasma and greatly damaged the optic nerves and tracts; the roots of the left olfactory tract were seriously damaged, and those of the right to a considerable extent. The hinder part of the tumour pressed against the inner edge of the left temporal lobe in the region of uncinate gyrus. Section showed the aneurism filled with firm, organised blood-clot, mostly laminated and pale, but in part black and more fluid. There was no rupture of its wall.

The aneurism was surrounded by fluid blood, and clots adhered to its right side and upper surface, and infiltrated

the finer membranes between the frontal lobes. These clots have now been removed from the specimen. Examination at the time showed that the left anterior cerebral artery crossed over the sac to its inner border so as to be connected with the right anterior cerebral, and then passed forward across the right side of the sac to reach the mesial surface of the left frontal lobe. The first part of the artery and the

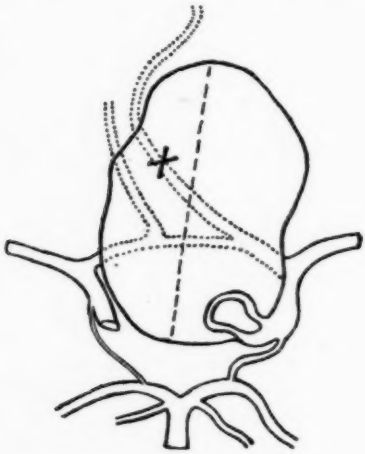


FIG. 2.

Diagram (natural size) of the aneurism of the left internal carotid, showing its relation to the adjacent arteries. The aneurism measured $1\frac{3}{4}$ ins. antero-posteriorly and $1\frac{1}{8}$ ins. transversely; it arose from the artery immediately that had pierced the dura mater. The anterior cerebral and anterior communicating arteries, indicated by dotted lines, pass above the sac, and the site of rupture of the left anterior cerebral is shown by the X. The broken straight line indicates the plane of section of the aneurism as seen in plate i., A. (Royal College of Surgeons Museum, 3795D.)

anterior communicating were embedded in firm clot over the sac. Just as the left anterior cerebral emerged from this clot, it was found torn across and surrounded by more recent clot (see fig. 2). There was no sign of atheroma of the arteries of the brain. The heart was not hypertrophied, but its ventricular walls were thin. The kidneys were slightly reduced in size, granular on the surface, and their capsules adherent.

The most pronounced feature of the case was a disorder of the senses of sight and hearing. This could readily be accounted for by the presence of the large intracranial tumour without supposing actual damage of the centres for these senses. Pressure of the large tumour on the optic nerve or even on the eyeball may have given rise to flashes of "bronze," the spectres, &c., while the hissing noise in the head might doubtless be the sound of the blood passing through the aneurism. The latter symptom is of particular interest. This remark was entered as a delusion on the reception order upon which the patient was certified as insane. She never complained of this noise after her admission to the asylum. For some years there were trivial signs suggesting some cerebral lesion, merely a slight degree of facial and third nerve paralysis, certainly not sufficient upon which to found a diagnosis of tumour. When, in the last few months of life, occasional vomiting not easily accounted for occurred, associated with a fuller development of the paralysis just mentioned, a diagnosis of tumour became more certain. Yet many of the most characteristic signs of a brain tumour never appeared. No headache, no pain in the head, no attacks of giddiness, no seizures or fits of any kind; and what is remarkable, considering the degenerated state in which the optic nerves were found, her eyesight remained good. To within a week or two of her death she was constantly employed at needlework.

It is impossible to account for the origin of the aneurism. There was not the slightest evidence of syphilis. She had several healthy children, and the husband appeared a healthy man. It may be mentioned, however, that a daughter of this patient had been an inmate of the asylum five and a half years before she herself was first brought there, but the daughter's mental condition appeared to have no relation to syphilis, neither did she show any evidence of that disease on her body. I mention this about the daughter, for, though not showing hereditary mental defect in the strict sense of the term, it yet proved there was doubtless some defect in brain development in the family.

How long the aneurism had been growing it is difficult

to estimate. There is no doubt that it had existed for over six years, and was, I believe, the cause of the patient's certification as insane; but it had probably been there in smaller size many years before. It is not often that a case of intracranial aneurism has been under almost daily observation for so long a period.

One more remark may be made on this case. The character of the clot around the aneurism pointed to the probability that bleeding from the ruptured artery occurred at more than one time; the first rush of blood, which formed the firm clot over the upper surface of the sac, was probably checked by the end of the vessel becoming blocked, followed later by further oozing from the torn vessel. This is not an isolated case. There is more than one aneurism in the museums pointing to rupture of the sac on at least two distinct occasions. As for the length of time between the attack of apoplexy and death of the patient, this has been greatly exceeded in some of the recorded cases.

This specimen ranks among the larger intracranial aneurisms on record. Its size has been exceeded by few, probably less than a dozen. Aneurismal tumours of very large size have generally occurred either on the internal carotid or on the basilar, but not invariably. Two of the largest vascular tumours on record have been described by Byrom Bramwell [7]; one was on the posterior cerebral, the other on the internal carotid. The first, which was the size of a small orange, was connected with the right posterior cerebral just after its junction with the posterior communicating—a very rare place for an aneurism. It occupied the whole space between the crura cerebri and optic tracts, filled the cavity of the third ventricle, and extended upwards into the lateral ventricle as well as protruding downwards at the base of the brain. There was naturally much displacement of the structures around the tumour; the optic nerves and chiasma were stretched, but not materially damaged. The walls of the sac were considerably calcified, but the arteries of the brain are said to have been almost free from atheroma. The heart was healthy, the kidneys cirrhotic. Cerebral symptoms had lasted for two and three-quarter

years, a little over a year of which was spent in Melrose Asylum. There was progressive dementia with loss of memory, and the patient, a woman aged 43, was subject to syncopal attacks. Three months before death the symptoms were suggestive of general paralysis, unsteadiness of gait, tremor of tongue, disorder of articulation, and general drowsiness. Two months later she could hardly stand; the left half of the face and the limbs of left side of the body were paretic; she suffered from headache, giddiness, enfeeblement of articulation and deglutition. Soon after she became helpless and bedridden; squint of left eye, pupils varied from time to time.

The second aneurism was an enormous oval cyst-like tumour measuring 3 ins. in its longest diameter, and was connected with the right internal carotid within the cranium. It caused a large erosion of the sella turcica; the orbital convolutions and neighbouring parts were flattened, the left optic nerve stretched and flattened over the tumour, the convolutions behind the Sylvian fissure flattened and the crura cerebri softened. The case was that of a man, aged 31, who died in Morningside Asylum. He first came under treatment, about six years before death, for failing sight of some months' duration; there was temporal hemianopsia of both sides, worse on the left. A year later this was complete, and there was marked atrophy of both optic nerves, followed a few months later by complete blindness, though the patient was otherwise in good health. Two years before death he was certified as insane with delusions of sight. There was now general weakness, but no paralysis; pupils dilated and insensible to light, objects appeared only as shadows. He sat all day with his head hanging down on his chest, and rarely spoke, though he could answer questions fairly well. No history is given for the last year. The aneurism had not ruptured. The condition of the cerebral arteries is not mentioned. The heart was fatty, the kidneys healthy, the genital organs atrophied or undeveloped.

The perimeter chart (fig. 3) shows the extent of loss of vision in the two eyes of this patient nearly three years before death. The only other case of intracranial aneurism

in which bitemporal hemianopsia has been mentioned was in the remarkable aneurism recorded by Weir Mitchell [41], and in this the chart is almost identical with that in Bramwell's case. The aneurism is supposed to have had its attachment to an abnormal artery passing across the optic chiasma, and both internal carotid arteries entered the sac. It formed a tumour larger than an egg, destroyed the optic commissure and caused great atrophy of both optic nerves and tracts, though more so of the left. It caused a large depression in the brain and dipped into the sella turcica. Except for headache, often severe, great sense of fatigue, and at one time slight weakness in the legs, his symptoms

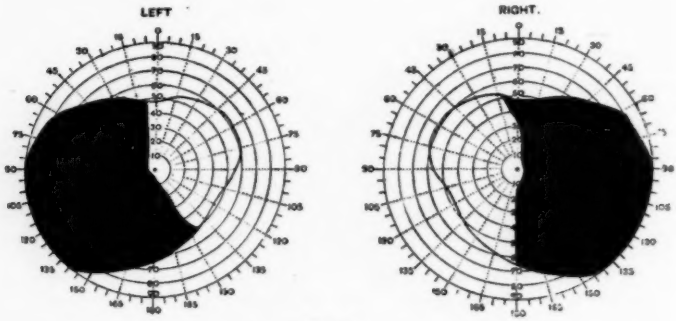



FIG. 3.

Perimeter chart showing bitemporal hemianopsia in a case of enormous aneurism of the right internal carotid artery, recorded by Byrom Bramwell, *Edin. Med. Journ.*, 1886-7, xxxii., p. 918. The chart was taken two years and eight months before the patient's death. From Bramwell's "Intracranial Tumours," 1888, fig. 27, p. 98.

appear to have been entirely connected with sight. These began about three and a half years before death with diminished vision in the left eye towards the left side. This increased, and six months later the vision of the right eye towards the right side began to fail, so that finally there was complete bitemporal hemianopsia. The fundus showed changes suggesting partial atrophy of the left optic nerve, and later on of the right also. He died suddenly after being in a comatose state a short time.

Notwithstanding the great destruction of the optic nerves and commissure that occurred in the patient I have mentioned, who died in Colney Hatch Asylum, there was no loss of vision recognised during life, so we must suppose that the changes seen in the nerve *post mortem* occurred within the last week of life; possibly much of the disintegration was effected after the blood extravasation took place. All we can conclude from the presence of temporal hemianopsia is that a lesion of some kind causes pressure on the central part of the optic chiasma. It is one of the most frequent symptoms in tumours of the pituitary body.

In Mitchell's case there is no mention of the patient seeing flashes of light, a phenomenon that Byrom Bramwell says [8] rarely occurs, except as a visual aura in epilepsy, in which it is usually followed by unconsciousness. In such cases, however, the subjective visual sensation is, as Bramwell says, the external manifestation of a discharge of grey matter in the cerebral cortex. In my case there was no indication of a lesion in the occipital region of the brain.

In the Middlesex Hospital Museum (No. 1101) is a large aneurism of the posterior cerebral, of which plate i., B., is a photograph. It is as large as a hen's egg, and lies embedded in the right cerebral hemisphere, having protruded up into the hinder part of the lateral ventricle. It was attached to a branch of the artery that passed into the ventricle with the velum interpositum. A large part of the temporo-sphenoidal lobe has been destroyed. The case was recorded by Van der Byl [61]. Three months before death the patient, a man, aged 56, was under treatment in St. Mary's Hospital for an attack of gout, but previous to this he had been an out-patient for pain in the head, vertigo, and debility of twelve months' duration. There were no head symptoms until eight days after admission, when headache and giddiness returned, which increased in the next week with severe pain at the vertex. He left the hospital after six weeks, complaining then of weakness only. About eight weeks later he was taken to Middlesex Hospital, having fallen down in the street in a fit, and died while in the waiting-room. The aneurism had ruptured.

A large globular intracranial aneurism, the size of a duck's egg, is referred to in the medical journals as having been shown at the Pathological Society of London by James Anderson in 1885 [3], but no account appears of this in the Transactions of the Society. It is said to have measured 2 by $2\frac{3}{8}$ ins., with circumferences of $5\frac{1}{2}$ and $6\frac{1}{4}$ ins. It had a well-defined capsule, and was almost completely filled with laminated clot, calcareous in places, and recent blood-clot at one part. It lay in the left middle fossa of the skull, and caused compression on the contiguous part of the brain,

FIG. 4.

A very large intracranial aneurism springing from the left middle cerebral artery, probably from Anderson's case. The artery has been laid open, and the dark circular spot indicates the small aperture between it and the aneurism. Natural size (as at present). Royal College of Surgeons Museum, 3795B.

with the fissure of Sylvius in the centre of the depressed area. This description exactly corresponds to the large aneurism, No. 3795B, in the Museum of this College, to which no history is attached (fig. 4). I have no doubt in my own mind that this is the aneurism referred to, and I may say that Mr. Shattock agrees with my suggestion. In the short account that is given in the medical journals, it is said that the specimen was found in a man aged 40 years, who had suffered at intervals from severe headache for four years, with some tenderness of the scalp. There were

paroxysms of vertigo, followed by twitchings (clonic spasms), and loss of power on the right side lasting for a few minutes. Slight right hemiplegia became permanent, and the right pupil larger than the left. Lowering of temperature on the side of the paresis was noticed. Double optic neuritis was present. Twitching of both legs, sudden and transient loss of power on right side, lasting but five minutes, with occasional involuntary micturition, vomiting, and slight exophthalmos. Death was preceded by vomiting, several fits, and coma.

This enormous mass is connected with quite a small artery, of which only a very small piece has been retained in the specimen. It is presumably the trunk of the middle cerebral. This is much the largest intracranial aneurism to be seen in any museum in London at the present time. Two specimens of aneurism of the cavernous portion of the internal carotid probably come next in size; one is the well-known case of Mr. Jonathan Hutchinson, now at the Royal College of Surgeons; the other is in St. Thomas's Hospital museum. I shall have occasion to refer to these again.

Two basilar aneurisms only of any size exist in the museums, and both are in that of King's College Hospital. To neither is there any history or reference. One, a fusiform aneurism about $1\frac{1}{2}$ ins. long and $\frac{3}{4}$ in. across, involving the lower two-thirds of the basilar, corresponds in every particular with an aneurism recorded by Kingston in 1842 [34], plate ii., A. For my own part I believe this is the specimen described; but even were it not so, we may reasonably believe that identical symptoms would have been produced. That occurred in a lad, aged 14, who was known to have had for the last three and a half years a large aneurism of the right internal carotid in the neck. He, however, seemed in good health, and there were no brain symptoms until bulbar paralysis suddenly appeared shortly before death. One morning on returning from work he was seen to tremble violently all down the right side of the body; he then vomited, and shortly afterwards lost power in the right arm and leg, showed paresis of right side of face, was unable to protrude his tongue; articulation was indistinct,

but mind not impaired. On the afternoon of the next day there was from time to time suspension of respiration for about half a minute, followed by spasm of larynx, then vomiting. He was unable to articulate, but understood what was said. He could not swallow either food or drink. There was now complete paralysis of the right side. Six hours later the breathing became stertorous; in the course of the next four hours he was totally insensible, and he died thirty-eight and a half hours after the onset of the attack.

The basilar artery was dilated into a large oval aneurism the size of a walnut, which was distended with firm clot, laminated in part, and there were coagula between its middle and outer tunics. The vertebral arteries entered the lower end of the sac about a line apart, and a short piece of basilar existed at the upper end. There was no rupture and no blood extravasation in the cranial cavity. Both sides of the pons were flattened by pressure, and considerably softened to a depth of three lines. The cardiac valves were normal.

The other is an unusually fine specimen of true sacculated aneurism of the basilar, on which artery aneurisms are more frequently of a fusiform character. It measures about $1\frac{1}{2}$ ins. across, and has its connexion with the artery by a small circular orifice about half-way up the vessel. The aneurismal sac lies across the left side of the pons, and the left fifth nerve is flattened over its surface. It is obvious that very marked and serious symptoms must have existed during life (fig. 5). Strange to say, this specimen is described in the old manuscript catalogue as a hæmorrhage into the pons. I pointed this out to Mr. Cheatle, and together we made a careful examination, with the result as stated. Owing to this incorrect diagnosis, it is not surprising that I have been unable to find the record of any case of which this is likely to be the specimen.

In plate ii., B., is seen a fairly large aneurism of the basilar (Royal College of Surgeons, No. 3795H.). It is from a man aged 68, who for twenty-one years had suffered from epilepsy following a fall from a horse. The last fourteen years of life were spent in lunatic asylums, owing chiefly to his occa-

sional attempts at suicide. Between these periods of acute depression the man was cheerful, a good worker, and showed very little impairment of faculties, although subject to fairly frequent fits, some of major and others of minor epilepsy. In the latter there was simply twitching of the facial muscles of the left side, and he did not fall. During the last six months there was some incoördination in both legs, and he dragged the left foot. There were never any symptoms on which to base a diagnosis of cerebral tumour; he never had headache or vomiting, and his eyesight was fairly good. There was only occasionally a little dizziness. He died

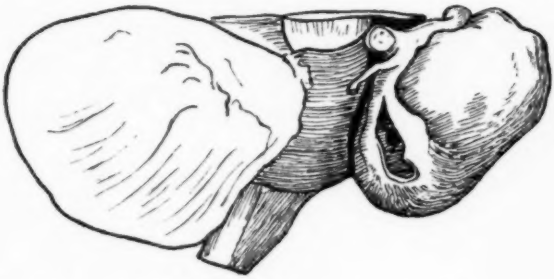


FIG. 5.

The right side of the pons and cerebellum, with the basilar artery, from which a large sacculated aneurism springs. The lower part of the artery is fusiformly dilated, and from the left side of its wall a circular orifice leads into the large aneurism that lies over the left half of the pons. The posterior cerebral artery is occluded by a firm thrombus. Natural size. King's College Hospital Museum.

suddenly from rupture of the sac. The aneurism measures nearly 3 cm. in length and 2 cm. in breadth, and lies mostly to the right of the mid-line, where its posterior wall is adherent to the superficial fibres of the pons.

In Squire's case [60] of considerable-sized aneurism growing forward from the first part of the left posterior cerebral (terminal division of the basilar), the patient died a few hours after coming under observation. There had previously been no paralysis of any kind, not even third-nerve symptoms (plate iii., A). In St. Thomas's Hospital Museum (No. 2052) there is an aneurism of almost the same size and form, but

its orifice opens directly into the anterior extremity of the basilar at its point of bifurcation. This aneurism measures ¾ in. in diameter, and covers the whole of the interpeduncular space. The case was published by Greenfield [22], who says that the patient, a male aged 16, was admitted to hospital with cardiac disease, and that there was an entire absence of cerebral symptoms (plate iii., B).

In the case of a sacculated aneurism, the size of a small hen's egg, long since recorded by Serres [56], the only sign before its final rupture was "weight and heaviness" in the head, to which the patient had long been subject.

When an aneurism has been situated at the upper end of the basilar there have sometimes been third-nerve symptoms. A good case of this has been given by Delpech [14], where the aneurism, the size of a pigeon's egg, protruded into the interpeduncular space.

An aneurism situated lower on the artery, though very much smaller, is, however, more likely to give rise to serious symptoms, owing to the proximity of the pyramidal tracts and the important cranial nerves. There may be paralysis, usually limited to one side. Difficulty in swallowing has been recorded several times. This was so in a case of Abercrombie's published in 1828 [1], where the basilar "for about an inch of its length was very much enlarged and hard, and this portion was completely filled up by a firm white matter without any appearance of blood." Again, respiratory embarrassment may be one of the most prominent symptoms produced by the tumour, as in one of Lebert's cases [36], where an aneurism twenty-one lines in length and fifteen lines across, partly compressed the medulla and effaced the pyramids. The vagus and pneumogastric nerves were slightly flattened, the roots of the hypoglossal partly destroyed, and the nerve itself atrophied. The patient lost all power of moving his limbs; there was difficulty of articulation, with gradually increasing loss of voice until it became entirely extinct, and death was preceded by increasing dyspnœa. Throughout the patient's intelligence remained fairly clear.

A good instance where nerve symptoms were produced is

that of Ruschenberger [53], where an aneurism the size of a pigeon's egg occurred at the lower end of the artery. The patient had attacks of choking and difficulty in swallowing even liquids, talked with difficulty, and had a complete paralysis of the left side. When sitting he required support, and there was a constant involuntary backward and forward vibration of the head and trunk and of the right foot while lying down, both when awake and asleep; the right hand was tremulous when used. Pfeufer's case [49] was very

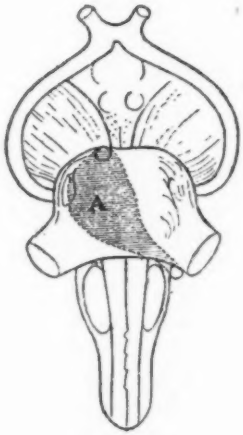



FIG. 6.

Diagram of the under surface of the mesencephalon, showing the site and size (indicated by the shaded area) of the aneurism of the basilar artery recorded by Hallopeau and Giraudeau, *L'Encéphale*, Paris, 1883, iii., fig. on p. 663.

similar, except for the involuntary movements referred to; instead, there was pain and stiffness across the spine and in the lower extremities.

In a man, aged 40, in whom the anterior half of the basilar was dilated to the size of a nut, causing grooving on the right side of the pons, recorded by Hallopeau and Giraudeau [25], a very remarkable phenomenon occurred towards the termination. When the patient was put into the sitting position, with the head bent forwards, respiration

was arrested in the stage of expiration, the pulse continuing to beat regularly for some seconds and then gradually becoming slower. If the head was then thrown backwards so as to extend the neck, the respiration was immediately re-established. These effects could be reproduced at will. The authors suppose that the phenomena observed were due to the pons becoming compressed by the aneurism when the head was flexed, whereas during extension of the neck the artery would be elongated and the aneurism carried out of reach of the pons (fig. 6). In this case the first symptoms to be noticed were girdle pains and heaviness in the limbs four months before death. These were followed by paraplegia, slowness of speech, incontinence of urine and fæces, dysphagia, convulsive movements of the right leg, acute pain at the back of the neck, gradual loss of consciousness deepening to coma, and finally death by gradual asphyxiation.

A correct diagnosis cannot always be made from the side on which the signs are most prominent or on which alone they are present. Ruschenberger's case is one in point. In that the symptoms suggested a lesion of the right side, but the aneurism was situated centrally. In Möser's case of dilated left vertebral, which will be referred to again, the diseased artery lay across the right pyramid of the medulla. In Schultze's case [55] of a sacculated aneurism on the left vertebral, practically the only symptom produced was spasmodic contraction of all the muscles of the left side of the face, except the frontalis. This existed for a year, and was increased by any movement of the jaws or face. The aneurism, as seen in his illustration, lies away from the pyramid.

The few aneurisms that have been observed on the various arteries of the cerebellum have generally been very small. The largest is that by Ogle [46], who believed the patient had a scrofulous tumour at the base of the brain. The aneurism, which is in St. George's Hospital Museum (No. 42L), is the size of a small nutmeg, and was situated on the left anterior cerebellar artery; it compressed the left crus cerebelli and contiguous parts of the pons, cerebellum, and root of the fifth nerve, while the seventh nerve was

stretched as it passed forwards (plate iii., c). The optic nerves, commissure, and tracts were softened and atrophied. The patient was subject to epileptic seizures for some years; there had been complete loss of sight for five years, followed by impairment of smell and taste on the left side with partial loss of muscular power on that side. Death occurred after convulsive attacks, in which the left side of the body was especially affected.

Vertigo and dizziness have been very inconstant signs in aneurisms at any part of the base. Headache is a symptom that has only occasionally appeared, though in a few cases it has been very intense. The position of pain in the head, when present, seems to bear no constant relation to the site of an aneurism. In a small basilar aneurism given by Gordon [20], frequent severe headache was present for a year, with weight over the temples.

Deafness has occasionally been a leading symptom. In a case of Loomis [38], deafness in the left ear came on two months before death: there was also numbness of the left side of the face, closely followed by paralysis of that side of the face and loss of taste. Otherwise the patient remained in good health until suddenly seized with vomiting; he showed partial loss of power in walking, and in a few days lost power of speech and died. An aneurism, apparently not ruptured, compressed the left side of medulla and pons. In one of Van der Byl's cases [62], the specimen of which is in the Middlesex Hospital Museum (No. 1102), the patient, between four and five years before her death, became suddenly and permanently deaf; but, as may be seen in the photograph (plate iv., A), the aneurism on the basilar lies quite away from the auditory nerve. The patient continued in her usual health, and went about until two years before death, when she had an apoplectic seizure. Two similar seizures occurred within the next two months, but she was able to walk about with assistance shortly after each attack. During the last six months there was gradually increasing helplessness. She much improved by rest in hospital, and was about to be discharged on the fourteenth day after admission, when she uttered a scream, fell down unconscious, and

died the next day. The aneurism, which had ruptured, has caused a slight depression over the centre of the pons. There is no mention of any sign of old cerebral hæmorrhage.

Taking the several arteries into consideration, we shall find that aneurisms of the basilar give rise to symptoms of tumour in a larger proportion of cases than aneurisms on any other artery at the base; this is not so much owing to the size of the aneurisms as to the fact that they cause compression on the motor tracts or on the roots of the cranial nerves, so many of which lie in such close proximity. In the whole series of collected cases, just half the patients with basilar aneurism had cerebral symptoms, apart from those of a final apoplexy. With aneurisms of the posterior communicating, 47 per cent. of the patients had symptoms, the most constant being such as are produced from compression of the third cranial nerve. But this nerve has often been involved when an aneurism is situated at the upper end of the basilar, or has taken its origin from the early part of the posterior cerebral artery. Aneurisms of the latter artery have been associated with symptoms in 41·6 per cent. of the cases, but in a number of these instances the symptoms have had little evident relation to the position of the aneurism. A remarkably large proportion of aneurisms of the posterior cerebral are of embolic origin, and generally occur on one of the smaller branches, where diagnostic symptoms are less likely to supervene. It may further be remarked that when such an aneurism occurs on one artery there is more often than not an embolic plugging of some other artery of the brain, and cerebral softening is usually found at more than one site.

In aneurisms of the middle cerebral arteries, symptoms occurred in rather less than 22 per cent. of the cases. These aneurisms, being placed on the artery at some distance from the large nerves, can only give rise to vague indications of their presence. Notwithstanding what is written in some books as to the possibility of diagnosing their presence, I do not believe that it is ever possible to do so. There is no evidence that any one has yet been diagnosed during life. The very most that can be said is that a guess may be

ventured that an embolic aneurism has formed on that artery when certain cerebral symptoms appear in a person already suffering from acute endocarditis. But such a conjecture would more frequently be wrong than right.

In aneurisms of the anterior cerebral and anterior communicating arteries, the number in which definite cerebral symptoms occurred is so trifling and so vague that most certainly such an aneurism is impossible of diagnosis.

In aneurisms of the intracranial portion of the internal carotid, definite symptoms occurred only in 31 per cent. of the cases. Naturally the nerve symptoms in these cases are associated principally with compression of the optic nerve, but the third nerve and others are sometimes involved. The third nerve may be compressed by quite small aneurisms of this artery, when their growth is in a backward direction, so as to occupy a position of an aneurism of the posterior communicating. This was the case in a specimen in the Middlesex Hospital Museum (No. 1100), of which an illustration is given (plate iv., B.). The sac is said to be the size of a hazel nut, and it presses against the exit of the third nerve. On the other hand, these aneurisms may reach a very large size without affecting that nerve. Another specimen in the same museum (No. 1097) shows an aneurism on the left internal carotid, which attained a diameter of about $\frac{3}{4}$ in. It has expanded mostly in an outward and upward direction, piercing the hinder part of the frontal lobe, and causing damage to the forepart of the corpus striatum. The optic nerve is flattened, and the roots of the olfactory tract absorbed. The body of the sphenoid is hollowed out (plate iv., C). The patient, a woman of 52, was admitted to hospital suffering from violent headache, with slight weakness and numbness of the right extremities. During the night she was suddenly seized with apoplectic symptoms, and died in a few hours. The aneurism ruptured into the lateral ventricle. The case is recorded by Moore [42], who gives no cause for the aneurism, but states that the arteries were healthy. The photograph (plate iv., D), from a specimen in University College Hospital Museum (No. 3475A), shows a thin-walled empty sac measuring over 2 cm. in

diameter. It occupies a similar position on the opposite side of the mid-line; it extends downwards instead of upwards; but, like the last mentioned, it presses on the optic nerve and trigonum olfactorium, which, with the adjacent cerebral substance, was softened. The rupture of the sac took place beneath the brain, instead of into it.

As one might almost expect, an aneurism of the internal carotid in its cavernous portion is of all those we are considering the most likely to give rise to definite symptoms. In my collected series, symptoms occurred in just under 69 per cent. of the cases. We have here symptoms produced by pressure on the several ocular nerves which pass through the sinus and on the ophthalmic division of the fifth cranial nerve. But even in this situation an aneurism may attain a considerable size without producing any very pronounced symptoms, while in other cases the symptoms have been comparatively slight and certainly not sufficient to locate the situation of a tumour, much less to permit a diagnosis of aneurism. In the specimen (No. 3226A) presented to the museum by A. H. Robinson, the aneurism was not suspected during life. The patient had divergent squint for some months and slight tremors of the right hand and arm; there was a sudden fit, coma, and death in a few hours. This large spherical sac, nearly an inch in diameter, is situated on the artery of the left side, at the side of the sella turcica, and caused depression in the temporo-sphenoidal lobe of the brain. There was no rupture. (plate v., A.)

In some cases of exceptionally large intracranial aneurisms the patients have developed mental symptoms, and there is no doubt that in these the aneurismal tumour was the cause of the insanity. On the other hand, as in most of my own cases, the small aneurism found accidentally in persons who died insane may probably be regarded as an accidental association. Yet in many of the latter class it is probable that the mental disease, like the aneurism, was the result of vascular degeneration of the cerebral arteries. Occasionally the mental disease has taken the form of general paralysis, or has so closely simulated that affection that during life the patient was looked upon as a general paralytic.

Occasionally the patients have been subject to epilepsy, either with or without definite insanity. In these cases the epileptic fits have rarely assumed a definite Jacksonian type.

Hæmorrhage into the retina has been seen in a few recorded cases of intracranial aneurism. It has generally occurred in association with meningeal hæmorrhage after the rupture of the aneurism, and may be accounted for by extravasated blood travelling along the sheath of the optic nerve. It would seem to give no help in determining the seat of an aneurism in the cranial cavity.

In an unpublished case of which I have the notes, subretinal hæmorrhages were seen. The patient was a man, aged 51, who had been an inmate of an asylum for ten years with delusional insanity. He suddenly had a seizure with loss of consciousness and general clonic movements. He then became dull and almost stuporose. Four days after the attack, ophthalmoscopic examination revealed several large hæmorrhages in the fundus of the left eye, and there was double optic neuritis. He complained of much headache; there was no paralysis, and he had no further fits. Four days later the temperature began to rise, he became drowsy and completely lost all power in the lower extremities, then passed into an unconscious state. Breathing became stertorous, and several times there was entire cessation of breathing for about thirty seconds. Death, ten days after the first attack, was preceded by pyrexia and a pulse rate of 150. Profuse meningeal hæmorrhage was found, caused by rupture of a small aneurism of the anterior cerebral artery, not larger than a pea.

A case very similar to this has been recorded by Mott and Stedman [44] as the result of rupture of a small aneurism of the anterior communicating artery, the blood extending along the sheaths of both optic nerves. The same occurred in a case under Hale White [65], the hæmorrhage coming from a small aneurism of the right internal carotid. In a small ruptured aneurism situated at the termination of the left internal carotid, recorded by Bramwell [9], both subvaginal sheaths surrounding the optic nerves were distended with blood and the optic discs were

swollen. Microscopically, inflammatory changes were found in the fibrous covering of the nerves and an excess of corpuscular elements in the trunk of the nerve itself. The heart and kidneys were healthy. Shortly before death the temperature rapidly rose to 107. It is not stated whether changes were observed in the retina during life.

In a case brought before the Ophthalmological Society by Hutchinson [31], where the basilar was the seat of an embolic aneurism that grew forwards into the interpeduncular space and compressed the optic commissure and nerves, there was sudden loss of sight twelve days before death. Examination of the eyes revealed recent hæmorrhages in both retinæ and general haziness of the fundi. In this case, the aneurism had not ruptured, and there was no meningeal hæmorrhage from other cause. The sheaths of both optic nerves were found distended with serous fluid and slightly infiltrated with leucocytes in the nerves behind the globes. Mr. Hutchinson suggested that this was the result of increased intracranial pressure through the tumour, but his view did not meet with general acceptance. Those who joined in the discussion expressed the opinion that the ocular and nerve changes were probably either a part of a general blood-infection from the septic endocarditis, or that the aneurism had set up a localised meningitis, with extension of the inflammation down the optic nerves.

An exceptional case of sub-conjunctival ecchymosis is recorded by Ross [52], where a small aneurism of the anterior communicating had ruptured in a youth, aged 17. Extravasation had spread to the right upper eyelid, the optic disc was engorged, and both eyeballs were very prominent. Hæmorrhage had extended along the sheaths of the optic nerves to the eyeballs. The cerebral arteries, apart from the aneurism, were healthy, and there was no heart disease.

It is stated by nearly all recent writers that cerebral aneurisms are capable of producing optic neuritis, though it is recognised that this sign, common with intracranial tumours from new tissue formation, is rare with aneurismal tumours. It is difficult to say how frequently th is does occur, for the eyes have been examined in a very small proportion

of cases. On the other hand, the rarity of symptoms suggestive of optic neuritis or atrophy points to infrequency of such changes. Optic neuritis is generally believed to be brought about by increase of intracranial pressure, or to direct pressure on the nerve. This, however, is not the explanation given by Gowers [21]. He regards "the origin of the papillitis in these cases as probably a descending inflammation extending to the nerve from that which already exists around an aneurism." This may perhaps be the explanation for such a case as Hutchinson's, but I doubt very much whether in cases of cerebral aneurisms neuritis is usually produced in this way. Aneurisms associated with an acute inflammatory state of the arterial wall, or in the adjacent tissues, form only a small proportion of those connected with cerebral arteries, and it may be noted that in practically all the cases where, ophthalmoscopically, retinal changes have been observed in proved cases of cerebral aneurism, the latter have been of large size, unassociated with any recognisable inflammatory process. The particular cases mentioned by Gowers as demonstrating the association of the optic nerve inflammation with aneurism are, to say the least, unfortunately selected. In one, namely, Jeaffreson's case [32], of supposed aneurism of the internal carotid in the cavernous sinus, there was no *post mortem*, although the patient died. In only one out of several cases said to have been recorded by Holmes, of Chicago, was there a *post-mortem* examination, and in that an aneurism of the internal carotid was found, but there was also an adjacent growth in the pituitary body. In Michel's case [39] double neuritis is said to have been the first sign of "a cirsoid aneurism of the two internal carotids," which pressed on the optic nerves, producing interstitial inflammation. Now, it so happens that Michel gives an illustration of this so-called cirsoid aneurism of the internal carotids. This is reproduced in plate iii., E, and I think it will generally be agreed that there is no evidence of aneurism of any kind. All you see is that the internal carotids, as they enter the cranial cavity, are perhaps a little large; it is a very common condition, and I can scarcely understand anyone describing it as an aneurism.

Double optic neuritis was present in Anderson's case of enormous aneurismal tumour which was apparently situated on the left middle cerebral artery. Atrophy of both optic nerves was present with the very large aneurism of the internal carotid recorded by Byrom Bramwell, but in the same author's case of a huge aneurismal tumour on the right posterior cerebral artery no examination of the eyes appears to have been made.

In these cases a very large tumour was present, which directly pressed on one of the optic nerves. But the aneurism in a few exceptional cases has not been so large. For instance, in the case of an aneurism the size of a cherry on the right internal carotid, recorded by Waldo [64], there was double optic neuritis. The case seems to have been uncomplicated by any other cerebral lesion, the arteries were healthy, and the cause of the aneurism is unknown. In a case recorded by Bruce and Drummond [10] we are told that there was at one time venous engorgement of the discs, not amounting to optic neuritis, and subsiding before death. An aneurism of the anterior communicating, the size of a small hazel nut, was found, but there was also a chronic basic meningitis. Indications of the latter lesion have rarely been seen in cases of cerebral aneurisms.

I have already referred to the fact that in the large aneurism of the internal carotid that was found in a woman who died in Colney Hatch Asylum there was the subjective symptom at one time of "a hissing noise in the head." This sensation seems to have been present for a very short period only—about five and a half years before the patient died, and about two years after the patient's mind was first noticed as being affected. Occurring in a lunatic, by whom the noise was attributed to electricity and who had typical auditory hallucinations, it was looked upon merely as an indication of her perverted mind, but I cannot help believing that it had some definite relation to the aneurism. At that time the sac would no doubt have been a more open cavity than it was found at death, and blood rushing into the sac would be liable to produce an aneurismal murmur, the sound of which would easily be audible to the patient.

Whether or not the murmur could be heard through a stethoscope I cannot say, for the patient never complained of this particular noise while in the asylum, and there was nothing to suggest auscultation of the head. It is almost strange that, if a murmur was produced in this case, it did not persist for a longer period (plate i., A).

In an aneurism of the anterior communicating artery (Royal College of Surgeons, No. 3795C), the size of a large cherry, with a diameter of 1·7 cm.—an exceptionally large aneurism for that particular artery—one of the chief points of interest is of a character very similar to that just mentioned in regard to the last case. The aneurism was found in a woman who died in the asylum, aged 63, and had been an inmate for six years. The aneurism has undergone a natural cure; it has a very dense, fibrous capsule, which has become calcified, and the sac is filled with firm, dark clot, with deposit of calcareous matter in the central parts. The mental state of the patient was that of subacute mania passing into dementia. No recognisable signs or symptoms were caused by the aneurism. Although the tumour comes into contact with the optic chiasma, as seen in this view of the base of the brain (plate i., C), it does not seem to have caused any noticeable compression, but some damage was caused to the inner and hinder angle of both orbital surfaces against which the tumour lay.

Whether the aneurism had any relation to the mental disease of the patient it is impossible to say. The patient had been insane for six years, and the aneurism, I should imagine, was probably of even longer duration. The point of more particular interest in the case is the fact that at the time of certification the patient, in addition to having delusions of suspicion, complained of queer feelings in her head like a "waterfall." She did not refer to this sensation after her immediate admission to the asylum. It is quite possible that at the time this remark was made the aneurism was an open sac in which blood freely passed in and out, and may have given rise to sounds which were misinterpreted by the patient.

In the case of the large aneurism of the basilar artery

from a man who died insane, there was no indication that at any time the patient heard sounds in his head. He did not even suffer from auditory hallucinations.

If you consult any text-book or treatise, either medical or surgical, that discusses the subject of intracranial aneurism at all fully, you will find that the writers invariably state that these aneurisms, in certain cases, give rise to an aneurismal murmur which is audible to the patient, and may be heard by others upon applying the ear to the patient's head. To the patient there is said to be a pulsating and throbbing sensation in the head, or a more definite sound may be heard; to the physician who applies the stethoscope there is a systolic murmur or bruit, usually to be heard with most distinctness in the temporal region. Some, like Epron [16], go so far as to state that an aneurism of the carotid artery within the cranium may be diagnosed with certainty if a murmur is perceived by the patient and by the observer, and arrest or diminution takes place on compression of the common carotid.

Now this statement appears to me to be one of those which we occasionally find copied from one book to another; originally resting on very slender evidence, but acquiring a spurious importance by constant repetition.

I doubt if more than a couple of living physicians or surgeons have seen a case in which a murmur has been audible, and in which the evidence is conclusive that the patient had a *true* aneurism of a cerebral artery. I say true aneurism, because we are not concerned with arterio-venous aneurism. No doubt exists that a bruit is probably always present when rupture of the internal carotid in the cavernous sinus has led to a communication between the artery and the vein. But the statement has been made of true aneurisms of the cerebral arteries.

There may be some who would doubt the truth of my remark. They might call my attention to the oft-quoted cases of Coe [12], Humble [29], Jeaffreson [32], Holmes of Chicago [28], and others, and to the numerous cases published in recent years, where a diagnosis of aneurism has been based largely on the presence of such a murmur, the

artery has been ligatured, and the patient frequently regarded as cured. In reply, I would point out that in none of these cases has the existence of an aneurism been proved. The histories of these cases always stop short at an interesting point. We are never told the sequel. In the few where we know that the patient subsequently died, there has been no *post-mortem* examination. I have read over the original reports of a vast number of cases. These include, besides the 555 proved cases of intracranial aneurisms which form the basis of my observations, a large number of cases—certainly more than a hundred, believed to be aneurism of one or other of the cerebral arteries—in which there was no autopsy, the greater part of those published under the heading of "Arterio-Venous Aneurism of the Internal Carotid," and cases published under the title of "Pulsating Exophthalmos" or an allied name; in short, all cases that could in any way bear upon this subject that I have obtained access to in the excellent library of this College.

As a result of this extensive reading, you may be surprised to hear that I have found two cases only proved after death to be uncomplicated true intracranial aneurisms, in which a murmur had been heard during life. One of these was an aneurism of the vertebral, the other an aneurism of the cavernous portion of the internal carotid.

The aneurism of the vertebral artery is recorded by Möser [43], and occurred in a man, aged 61, who suffered from long-standing mitral disease. A number of interesting symptoms, indicative of bulbar lesion, had been present for a considerable period. Four days before death *a loud murmur was heard on each side between the mastoid process and the vertebral column*, that on the right side being the loudest. On examination after death the left vertebral artery showed an aneurismal dilatation, causing a shallow but appreciable depression on the anterior surface of the medulla. The dilated part of the vessel was 12 mm. wide and 20 mm. long. The basilar was also strikingly wide, and contained soft fresh clot. The intima showed slight fatty degeneration. A section through the medulla showed considerable diminution of the restiform body and flattening of the right olivary body.

There were foci of softening in the corpus striatum and thalamus of each cerebral hemisphere. No abnormality was seen in the pons and crura. The left carotid artery was somewhat dilated; it contained blood-clot, but no embolus.

Möser illustrates this so-called aneurism, which is reproduced as fig. 7. The interesting and very pronounced symptoms were caused, as you see, by a comparatively slight dilatation of the vertebral artery, which lies across the opposite side of the bulb to which the vessel rightly belongs. With the record of this case, Möser gives two more

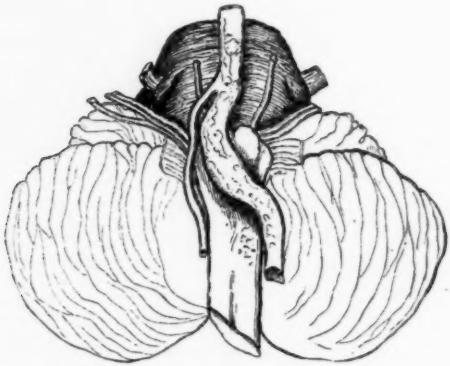


FIG. 7.

Möser's case of aneurism of the left vertebral artery, which was associated during life with a loud murmur heard between each mastoid process and the vertebral column. From *Deut. Arch. f. Klin. Med.*, 1884, xxxv., fig. on p. 441.

cases of aneurisms of the vertebral artery, proved by *post-mortem* examination. In the one the dilatation was as wide, in the other even greater than that just mentioned, but in neither case is there any mention of a murmur being heard. He also mentions a case which occurred in the private practice of Prof. Gerhardt, in which, with symptoms suggesting a lesion pressing on the medulla, a systolic bruit could be heard between the mastoid process and the vertebral column on the left side only. Möser's interpretation was either the gradual formation of an aneurism of the left vertebral or

partial blocking of that artery by an embolus, but there was no *post mortem* to prove the correctness of either suggestion.

The aneurism of the internal carotid is Hutchinson's well-known case [30], of which the specimen is now in this Museum (No. 3226). The patient, a woman, first came under observation at the age of 40 for eye symptoms, complete paralysis of the left sixth, incomplete paralysis of all branches of the third, and probably ocular branches of the vaso-motor. Dimness of sight and drooping of the eyelid had existed for about a year, but she had been liable to bad headaches for over ten years, associated for the last twelve months with "dreadful throbbing" in the temple and with a sensation of "beating under the ears," which lasted for two or three days at a time and prevented sleep. During the course of the next year the paralysis of the cranial nerves increased and the fifth became implicated, for she complained of numbness and tingling feeling in the left cheek, side of tongue, jaw and eyelid. About this time a bruit was detected, and Mr. Hutchinson says: "My diagnosis of aneurism was so confident that we had on one occasion fixed the date for the ligature of the carotid." The patient, however, refused to undergo an operation. The symptom of throbbing slowly subsided, but the ptosis and paralysis of the recti muscles remained complete. She improved somewhat in health. Eyesight was little if at all affected; there is no mention of exophthalmos. Death occurred eleven years later. It was caused by bone disease and abscess produced by a large dissecting aneurism of the abdominal aorta that had been recognised about a year. The aneurismal tumour on the left internal carotid, on its cavernous part, is the size of a bantam's egg; it rests on the Gasserian ganglion, which is greatly flattened; the motor nerves of the eyeball are stretched over its wall; the optic nerve is in close apposition, but shows no sign of compression. The tumour had its opening from the artery by a small circular aperture, and was filled with organised clot (plate v., B). This case has frequently been quoted by subsequent writers as an instance of spontaneously cured intracranial aneurism.

Three cases of pulsating exophthalmos, with the asso-

ciated phenomenon of a murmur in the head, are often referred to as due to the rupture of true aneurisms of the internal carotid into the cavernous sinus. These are the cases of Nunneley [45], Baron [4], and Gendrin [18]. But a study of the original reports proves that in none of these cases was there conclusive evidence of a true aneurism of the carotid artery.

I know of only two cases of true aneurism of the internal carotid associated with pulsating exophthalmos, but both were complicated with other conditions that might have accounted for the murmur. One, a published case, was reported at a later date than the chief papers dealing with pulsating exophthalmos; the other is unpublished.

Dempsey [15], under the title of "orbital aneurism," records a case in which there was a definite aneurismal dilatation of the internal carotid artery in its cavernous part, associated with the usual symptoms during life of pulsating exophthalmos. After confinement of her first child, the patient, a woman, aged 22, slept for some hours, and on awaking had headache and heard slight noises and singing in the head. At breakfast she had an attack of vomiting and "felt a sudden rush of something coming up from the left side of her neck and chest and settling in her eye with a 'snap.' This was immediately followed by intense pain in the eye and head, and numbness of the affected side. She became faint and was unable to articulate for some time." The eye became swollen and inflamed, and then gradually protruded. When seen two months afterwards she had total loss of vision of that eye, and there was a purulent discharge from under the lids. There was a distinct pulsation and vibratory thrill over the globe, most marked at the inner angle of the orbit. A loud whizzing bruit, synchronous with the cardiac systole, was heard over the orbital arch and forehead, less distinctly over the temple and malar bone, and was immediately and completely arrested by compression of the left common carotid. She complained of loud noises in the head and eye which prevented sleep at night.

She was treated by rest in bed, a low diet, and iodide of

potassium, together with digital compression of the common carotid artery. Slight improvement followed. A month later the patient experienced the sensation of a sudden rush into the left ear, accompanied by a crack similar to what had occurred in the eye. She became faint with intense pain and deafness of the left ear. A loud bruit was now heard above the ear. Pain became more severe and constant, noises louder, and general symptoms more alarming. About a fortnight after this second attack, the common carotid was ligatured. Pulsation and thrill, though much diminished, were not arrested, the eye became gradually less prominent and chemosis partly subsided, mobility of the globe returned, she could close the eyelids and the bruit became faint. The patient progressed favourably for three weeks, when secondary hæmorrhage occurred and the vessel was again ligatured. The eye symptoms returned and the globe again protruded. Three weeks after the second ligature was applied, violent hæmorrhage took place through the ulcerated cornea; it recurred two days later and the patient died.

There was a fusiform aneurism of the left internal carotid by the side of the sella turcica throughout the cavernous sinus, involving the origin of the ophthalmic artery. It was slightly sacculated by the side of the body of the sphenoid, and measured $\frac{1}{2}$ to $\frac{3}{4}$ in. across its widest part. There was no communication with the sinus. The ophthalmic artery from its origin was dilated to four times its natural size, and opened into an aneurism in the orbit which could easily contain a mandarin orange; the centre of the cavity contained recent clot with firm laminated old clot around, except anteriorly, where a communication existed with the eyeball. The arteries of the circle of Willis were all dilated, especially the anterior communicating, and the veins of the orbit were all very much enlarged.

Here there was a true aneurism of the internal carotid artery, but, as you see, it was so much complicated by other conditions of the ophthalmic artery that it is impossible to regard the murmur and other symptoms present as due to the dilatation of the carotid artery.

The unpublished case was under the care of Mr. Haynes

Walton, and the specimen is in the Museum of St. Mary's Hospital (No. 701). It shows two aneurismal dilatations of the internal carotid; the upper one, from which the ophthalmic branch springs, was in the cavernous sinus, the lower one was just outside the skull. Between these the carotid itself seems dilated. The ophthalmic artery and its branches are greatly dilated, constituting a cirsoid aneurism (plate v., c). It was from a man in whom symptoms appeared soon after receiving a kick on the head from a horse. There was a rough systolic bruit over the frontal sinus, proptosis and pulsation. The common carotid was tied, but pulsation returned in the orbit a few hours after the operation. The patient did well for three weeks, when he developed a high temperature and died. Congestion without softening of the brain was found. The specimen is regarded as an instance of aneurisms, one of them cirsoid, due directly to injury.

In this case, again, it is impossible to assign the murmur heard in the head to the dilatation of the internal carotid, owing to the fact that associated with it there was an aneurismal state of the ophthalmic artery, which of itself would probably have caused pressure on the cavernous sinus and veins of the orbit.

Against these we may set a large number of cases where there has been no evidence of the existence of an aneurismal murmur. In some, where the aneurism has been much larger, we are definitely told that there was no noise in the head, and in a few instances that there was no murmur to be heard on auscultation. In not one of the large aneurisms of the basilar is there any distinct evidence of its existence. Aneurisms of the vertebral of a truer form have much exceeded that of Möser's case without giving rise to any sound. There is not a single case on record of a murmur having been heard in proved cases of aneurism of the true *intracranial* portion of the carotid, or on any of the other larger arteries at the base of the brain.

Dr. A. H. Robinson tells us that in his case of a spherical sac, an inch in diameter, on the internal carotid in the cavernous sinus there was nothing to indicate the presence of an aneurism. The ophthalmic artery, which is dissected

out in the specimen (No. 3226A), is pervious and not enlarged (plate v., A). In Gilbert Blane's case [5] of aneurisms on both internal carotids in the cavernous sinus, which are in this Museum (Nos. 3223 and 3224)—the only case of symmetrical aneurisms at this site on record—there is no mention of a sound being heard by the patient. There is nothing of the kind mentioned by Holmes [27] for the specimen preserved in St. George's Hospital Museum (No. 42A): an aneurism the size of a walnut on the left internal carotid in the cavernous sinus, of embolic origin, in a boy aged 16. The same remark applies to an equally large aneurism in the same situation, but on the artery of the right side, recorded by Adams [2]. No mention is made of noises or murmur by Czermak [13] when recording an aneurism of the left internal carotid, from the anterior part of which the ophthalmic branch arose. This aneurism formed a tumour 5½ cm. in diameter in the middle fossa of the skull, and was mostly covered by the dura; it compressed the fifth nerve and Gasserian ganglion, stretched the third and fourth nerves, and caused a depression with softening of the under surface of the temporal lobe of the brain. The symptoms were those one would naturally expect from such a lesion, and persisted for about eight years. Death was due to apoplexy from rupture of the sac—practically the only case of rupture of an aneurism on this part of the artery.

In St. Thomas's Hospital Museum is a remarkable specimen (No. 1685), the particulars of which, Mr. Nettleship tells me, have never been published. The patient, a woman aged 61, came under the notice of Mr. Nettleship on July 5, 1889, when symptoms had already existed for eighteen years. These began with pain in the nose, followed in a few weeks by drooping of the left upper eyelid and protrusion of the eyeball, which was turned outwards. The ptosis diminished after six months, but never wholly disappeared; the eye gradually turned in and remained so for the next ten years. Two and a half years before being seen, the patient lost the power of swallowing, fluids returned through the nose, and the projection of the eye, which had subsided, again returned.

Dropping of blood from the left nostril was now observed, followed by profuse bleeding, which recurred several times during the next three months. She kept her room six months, and the eye again receded. In January, 1889, an attack of sudden bleeding again took place, which was repeated at about monthly periods until June 11, when she lost nearly a quart of blood.

When seen by Mr. Nettleship, both nostrils were quite free. There was semi-ptosis and complete paralysis of the sixth and third nerves, and possibly of the fourth. Left optic nerve atrophied, retinal vessels somewhat shrunken. Extreme internal strabismus, doubtful slight proptosis. Condition of fundus of right eye not mentioned, but she saw well with that eye. For five years there had been deafness of the left ear. There was no noise or beating in the head. For a year past she had noticed profuse sweating of the left side of the head and face, possibly from the involvement of the cervical sympathetic by a small aneurism which existed at the root of the neck. No history of injury, but a probable one of syphilis, for she had had many miscarriages and had been twice married.

On July 10 the patient lost 16 ozs. of blood while in the hospital. Mr. Sydney Jones, to whose ward the patient was transferred, had decided to tie the left common carotid on August 1 at 3 p.m., but about noon of that day sudden hæmorrhage came on and the patient died in five minutes, in spite of pressure being made over the artery.

An examination of the head only was permitted after death. An aneurism of the cavernous portion of the left internal carotid formed a large tumour $1\frac{1}{2}$ ins. in diameter, projecting at the side of the sphenoid into the middle fossa of the skull (plate v., D). The aneurism was partly filled with laminated clot, and between it and the naso-pharynx was an irregular communication. The left optic nerve was displaced outwards, and the third nerve was pushed outwards and backwards; the fourth and sixth nerves were also displaced.

This aneurism closely resembles Mr. Hutchinson's, except that it is larger, and there is the communication with

the naso-pharynx. It is difficult to explain why in the one there was a loud sound produced and not in the other, unless this was owing to the artificial channel that had formed, which enabled blood to escape from the sac.

In this last case, the Gasserian ganglion would seem to have escaped the pressure to which it is usually subjected by large aneurisms in this situation. In a case recorded by Romberg [51], intense neuralgia was a most prominent symptom, due to irritation of the Gasserian ganglion by a rigid and somewhat dilated internal carotid artery in the sinus.

A sensation of intense buzzing in the ears, existing for some time, occurred in cases of aneurisms of the intracranial carotid recorded by Boudet [6] and by Lindner [37]; in the former the sound is said to have been intermittent. In Shaw's case [57], in which there was an aneurism on the right internal carotid the size of a walnut and another on the right middle cerebral the size of a pea, the patient suffered from a buzzing sound and acute pain in the right side of the head; she was a woman, aged 50, who died insane. There was headache for two years, with a feeling as of the blow of a hammer over the right orbit, in a case recorded by Giraudet [19], in which there was proved to be an aneurism of the right internal carotid arising at the origin of the ophthalmic artery. This aneurism was situated above the cavernous sinus; the sinus was obliterated and the clinoid processes absorbed. In Stumpff's case [59], and in a few others, severe throbbing pains in the head have been noticed. In the case of a large aneurism of the left posterior cerebral artery recorded by Hanot [26], persistent singing in the ears occurred. The sac filled up the inter-peduncular space and projected into the third ventricle. In Bramwell's large aneurismal tumour in this same situation, there is no mention of any noise or singing in the ears, and no suggestion that any sound was heard by the patient. In Chevalier's case [11], in which a spherical tumour as large as a walnut filled the sella turcica—believed to be an aneurism, but its arterial connection was not found—the patient felt a shaking or beating sensation in the head.

In an aneurism of the basilar given by Pfeufer [49], there was constant ringing in the ears, but this was after partial recovery from an apoplectic attack. In Guéniot's case [23], where both the basilar and left vertebral were considerably dilated in a diffuse manner, there was also singing in the ears. The same sensation was experienced in a case given by Varrentrapp [63], where a limited dilatation of the basilar the size of a pea was found in front of the pons.

I have failed to find a single case of aneurism of any artery of the brain in which a patient has complained of a "hissing noise" or a sound "like that of a waterfall," such as were experienced in my own cases. The nearest approach to these was in a case recorded by Klippel and Boeteau [35], where the patient said he felt a bubbling in the head and heard a continual roaring sound. There was a large sacculated aneurism of the basilar which compressed the left half of the pons, and its upper extremity reached the left crus cerebri and compressed the optic tract (plate iii., D). It occurred in a man, aged 35 years, whose symptoms began two years before death with sudden intense headache, when apparently in perfect health. At first it appeared at the nape of the neck, but soon travelled to the top of the head. Heaviness and stiffness in the legs supervened and walking became difficult. Muscular stiffness extended to the upper limbs. From time to time there were spasms of a temporary character. Speech became slow, vision defective, ending in blindness. Persistent dysphagia and pharyngeal anæsthesia were present, and the velum palati was paralysed. Epileptiform attacks the last three months, with weakness of intellect, then habitual somnolence; finally sudden death from rupture of the sac. The arteries of the brain were healthy, and there was no history of rheumatism, syphilis, or alcoholism, though his father was an alcoholic and his mother died of cerebral softening after showing signs of insanity. The authors remark on the fact that none of the usual causes of aneurism were present, and add: "There exists, therefore, a group of aneurisms whose etiology is unknown." I may add that an intracranial tumour was diagnosed, but not an aneurism. The patient had been certified as insane.

There have thus been about a dozen cases recorded in which some more or less definite sound has been heard in the head by the patient, but in which apparently no auscultation of the head was made by the physician under whose observation the patient came. The sounds in most of these cases, such as buzzing, ringing and singing in the ears, might be due to causes very different from an aneurism of the cerebral arteries, and would, I fear, be of little value for diagnostic purposes, though, taken with the other symptoms present, they should lead one to apply a stethoscope to the head for the detection of a possible brain murmur.

Having shown that in true aneurisms within the skull a murmur is rarely produced, I would point out that aneurismal-like murmurs have been heard during life in fairly numerous cases where the absence of aneurism has been proved on *post-mortem* examination. In these cases there has usually been some entirely different lesion of the brain. Sometimes on the strength of the murmur an incorrect diagnosis has been made, and in some cases the carotid has been ligatured in the neck with a view to curing the supposed aneurism.

Mills [40] mentions a case in which an aneurism was diagnosed owing to the presence of pulsation and a bruit, both of which were lessened by pressure on the carotid arteries, but which at the autopsy proved to be a case of gliomatous tumour causing erosion of the skull. Vascular murmurs have also been heard in cases of extensive softening. Such a case has been recorded by Allen Starr [58].

Oppenheim [47], it may be mentioned, does not consider murmurs in the head a safe sign of aneurism. Writing on this subject, he points out that a pulsating vascular murmur may be due to tumours which arise in the vicinity of a large artery and compress it, or to very vascular tumours. Cephalic murmurs, he says, have been detected several times in exophthalmic goitre, and as a result of compression of the sympathetic nerve by tumours. He also states that similar sounds can be perceived on auscultation of the skull in cases of extreme anæmia in the adult, but this statement, Sachs [54] considers, requires further corroboration. It has been

pointed out by several writers that in childhood these murmurs are of no pathognomonic importance, for they occur in rickets, anæmia, chronic hydrocephalus, and even in perfectly healthy children. Osler [48] dealt with this subject very fully some years ago, and to his interesting paper I would refer you for the several suggestions put forward to explain the systolic brain murmurs in children. In the adult a similar murmur has been attributed to a congenital narrowness of the carotid foramen [47].

The group of symptoms which make up the condition now usually known as pulsating exophthalmos, in which there is practically always a systolic murmur to be heard over the orbit and sides of the head, may be regarded as proved to be caused by obstruction to the backward flow of blood from the ophthalmic vein. The causes that give rise to this obstruction have been shown to be very various, but as a general rule there has been pressure from some cause or other on the cavernous sinus. A rupture of the internal carotid artery in the sinus is doubtless one of the causes, by producing an aneurismal varix. It is extremely doubtful if a dilatation of the artery alone is sufficient. How the murmur is produced in these cases has not, I believe, been satisfactorily explained. In a few cases of exophthalmos, with bruit and pulsation, examined after death, nothing has been found to which the condition could be attributed; such a case is published by C. Williams [66].

In further confirmation of my statement that it is doubtful whether true aneurisms of the intracranial arteries are associated with an aneurismal murmur, except in very rare instances, I may mention that Sir Victor Horsley, who has made auscultation of the head a routine practice in his examination of cerebral cases, gives me permission to say that in the whole of his experience he has never met with any instance of a murmur being heard over the head in any case in which the presence of an intracranial true aneurism has been definitely proved by operation or by *post-mortem* examination.

When opening a discussion on "Intracranial Lesions" before the New York State Medical Association in October,

1890, W. W. Keen [33] briefly referred to a case in which Horsley, by lifting up the frontal lobes of the brain, exposed what was believed to be an aneurism at the base pressing on the optic chiasma, for which he ligatured both carotid arteries. This case has never been more fully reported, but Sir Victor Horsley has kindly given me the following particulars: At the operation a large cystic tumour, in close proximity to the internal carotid artery, was seen; a diagnosis of aneurism was made, and was believed to be confirmed by the fact that a loud systolic murmur was heard at the side of the head in the temporal region, and an impulse was felt in the tumour. The patient died some years afterwards, and the supposed aneurism proved to be a large blood-cyst connected with the anterior part of the pituitary gland. It seems clear that in this case the pulsation was transmitted to the tumour from the adjacent carotid arteries, and the murmur may possibly be explained by pressure of the tumour upon one or both of the arteries.

On the other hand, Sir Victor has given me an account of a case which there were good grounds for believing to have been one of intracranial true aneurism, in which no murmur was heard, even on applying the stethoscope directly to the tumour itself. He was called upon to operate upon a woman, aged about 48, with symptoms pointing to a tumour in the middle fossa of the skull. She had signs of external ophthalmoplegia—complete paralysis of sixth nerve, partial of third, and pain in region supplied by the middle division of the fifth. No murmur was detected by Horsley on auscultation of the head. On examining the under surface of the brain at the operation, a large pulsating tumour, having the characteristic purplish colour of the arteries, was seen; it measured at least $1\frac{3}{4}$ ins. in diameter, and sprang from the right internal carotid immediately after that artery pierced the dura mater. The tumour was cystic to the touch, and on applying a sterilised stethoscope to its surface not the slightest sound or murmur was audible. The right common carotid was ligatured in the neck; and now, about five years since the operation, the patient is in extremely good health.

Notwithstanding the remarks made by some who have

specially written on intracranial aneurisms and the writers of some of our leading text-books, the conclusion that I have been forced to draw from a careful study of a large series of cases is, therefore, that it is quite impossible to diagnose an aneurism of any one of the cerebral arteries except in the most unusual circumstances. Only two or three have ever been diagnosed during life, and even in these cases it can scarcely be said to have been an absolutely certain diagnosis. Hutchinson [30] even remarks as regards his case: "The chief question of diagnosis was between aneurism and pulsating tumour."

I will go further and say that in the vast majority of cases of aneurism a tumour even cannot be diagnosed. I have mentioned a number of interesting cases, which presented symptoms, one might say, clearly pointing to an intracranial tumour. But these, I would point out, were exceptional cases. They are in the minority. With the majority there have been no distinguishing signs or symptoms. Usually when they occurred they might readily be produced by other lesions than those of a tumour. In many cases there has been an entire absence of symptoms, sometimes when the aneurism has been of considerable size. The apoplectic symptoms following rupture of an aneurismal sac are indistinguishable from those of cerebral or meningeal hæmorrhage from other causes.

If there is any one sign to which special attention might be drawn, it is the occasional intermittent character of the symptoms. But these form a very small proportion even of the cases where tumour symptoms were present, and, after all, it is not very uncommon for symptoms to show variability in intensity, or even a temporary disappearance, in some other forms of tumour and in other cerebral diseases.

This impossibility of diagnosing a cerebral aneurism by those who have recorded cases cannot be placed to the ignorance of the observers. Many of the cases have been under the direct observation of the most learned physicians of the day, both in this country and abroad. The exact diagnosis of a cerebral aneurism during life is no more possible to-day than it was when Gull wrote nearly fifty years ago [24]: "Although we may from the circumstances

sometimes suspect the presence of aneurism within the cranium, we have, at the least, no symptoms upon which to ground more than a possible diagnosis."

## Description of Plates.

### Plate I.

(A) Aneurism of the intracranial portion of the left internal carotid artery. The anterior portion of the left cerebral hemisphere, viewed from the mesial surface, and showing the aneurism in section. The sac is filled with laminated clot except for a narrow cleft which held recent coagulum and fluid blood. The clots around the sac have been removed. From Ballance's "Some Points in the Surgery of the Brain," 1907, fig. 72, p. 167. Royal College of Surgeons Museum, 3795D.

(B) A part of the under surface of the brain, showing a large aneurismal tumour embedded in the right cerebral hemisphere and lateral ventricle. The aneurism is the size of a small hen's egg and is entirely filled with laminated clot. It is attached to a branch of the posterior cerebral artery which passes into the ventricle. (Van der Byl.) Middlesex Hospital Museum, 1101.

(C) Aneurism of the anterior communicating artery. The aneurism, with hard fibrous walls, forms a small tumour the size of a cherry in the midline at the base of the brain, immediately in front of the optic commissure, which is slightly pushed back. Six years before death the patient complained of hearing sounds in the head "like a waterfall." The left half of this aneurism is shown in the specimen. Royal College of Surgeons Museum, 3795C.

### Plate II.

Two large fusiform aneurisms of the basilar artery. Natural size.

(A) An aneurism of the lower two-thirds of the basilar about 1¼ ins. long and ¾ in. across, probably from the case recorded by Kingston, *Edin. Med. and Surg. Journ.*, 1842, lvii., 69. An incision has been made through the length of the aneurism and shows its interior filled with firm laminated clot. The vertebrales enter the lower end of the sac about half an inch apart. The basilar artery immediately above the sac has apparently been divided artificially. King's College Hospital Museum.

(B) Aneurism of the central part of the basilar, measuring 3 cm. in length and 2 cm. in breadth. The sac is empty and is ruptured on its anterior surface. Royal College of Surgeons Museum, 3795H.

### Plate III.

(A) Large aneurism arising from the first part of the left posterior cerebral artery and filling the interpeduncular space, which caused sudden death without previous cerebral symptoms. There are several anterior communicating arteries and the basilar is extremely small. The third nerves are adhering to the hinder part of the tumour. Squire, *Trans. Path. Soc.*, 1856-7, viii., fig. 8 on p. 167.

(B) An aneurism, ¾ in. in diameter, arising from the upper extremity of the basilar artery and completely covering the space within the circle of Willis. It occurred in a youth aged 16, suffering from cardiac disease, and there was an entire absence of cerebral symptoms. Greenfield, *Trans. Path. Soc.*, 1877-8, xxix., 91. St. Thomas's Hospital Museum, 2052.

(C) Aneurism of the left anterior cerebellar artery, the size of a nutmeg, resting upon the middle crus cerebelli, the contiguous parts of the pons and cerebellum, and the apparent origin of the fifth nerve. Ogle, *Med. Chir. Trans.*, 1859, xlii., plate vi. (reduced)

(D) Large sacculated aneurism of the basilar artery recorded by Klippel and Boeteau. During life the patient heard bubbling and roaring sounds in the head. *Bull. Soc. anat. de Paris*, 1892, lxvii., fig. on p. 86 (reduced).

(E) Michel's so-called "cirsoid aneurism of the two internal carotids," which are said to have pressed on the optic nerves, producing interstitial inflammation. Reproduced from *Graefe's Arch. f. Ophth.*, 1877, xxiii., 2, Taf. i., fig. 4, facing p. 254.

PLATE IV.

(A) Aneurism of the central part of the basilar artery, $\frac{3}{4}$ in. in diameter, associated with deafness appearing suddenly five years before death. There was atrophy of both auditory nerves, believed to have resulted from pressure of the aneurism on small auditory branches of the basilar artery. (Van der Byl.) Middlesex Hospital Museum, 1102.

(B) Aneurism of the right internal carotid, the size of a hazel nut, growing backwards and pressing on the third nerve. Middlesex Hospital Museum, 1100.

(C) Aneurism of the left internal carotid, globular in shape and about $\frac{3}{4}$ in. in diameter. It has grown mostly in an outward and upward direction, become embedded in the hinder part of the frontal lobe and passed through the anterior horn of the lateral ventricle, into which it had ruptured. (Moore.) Middlesex Hospital Museum, 1097.

(D) Thin-walled aneurism of the right internal carotid. The globular-shaped aneurism, measuring 2·2 cm. in diameter, arises at the bifurcation of the artery into the middle and anterior cerebrals. It has grown downwards and inwards, and, rupturing on its under surface, caused blood extravasation into the finer membranes at the base of the brain. University College Hospital Museum, 3475A.

PLATE V.

Aneurisms of the cavernous portion of the internal carotid artery. Natural size.

(A) Aneurism of the left internal carotid artery in its cavernous portion, viewed from above. (Robinson.) The specimen shows the left orbital cavity from which the roof has been removed, and its contents removed except the hinder part of the eyeball, a small part of the optic nerve, and the ophthalmic artery. Behind the orbit, at the inner side of the middle fossa of the skull, is the globular sac of the aneurism, having a diameter of a little over 1 in. The dura mater has been removed. The internal carotid artery is divided just above the level at which the ophthalmic branch is given off. The sac and artery communicated by a small circular orifice at the last upward bend of the latter. Royal College of Surgeons Museum, 3226A.

(B) Aneurism of the left internal carotid in its cavernous portion, in which a bruit was detected eleven years before death, recorded by Hutchinson, *Trans. Clin. Soc.*, 1875, viii., p. 127. The inner side of the aneurism, with the artery laid open in its passage through the carotid canal and cavernous sinus, and showing a small circular opening leading into the sac. Royal College of Surgeons Museum, 3226.

(C) The upper part of an internal carotid artery with two aneurismal dilatations, the lower one just before the artery entered the carotid foramen, and the upper larger one on the artery while in the cavernous sinus. The ophthalmic artery and its branches are much dilated, constituting a cirsoid aneurism. St. Mary's Hospital Museum, 701.

(D) Large aneurism of the left internal carotid artery in its cavernous portion. (Nettleship.) The part of the base of the skull forming the left middle fossa is shown, in which the aneurism lies. The left internal carotid has been laid open (except for a narrow band of dura mater over the top of the sinus) and shows a considerable-sized opening leading into the sac. A bristle is passed through the right internal carotid. St. Thomas's Hospital Museum, 1685.

PLATE I.

A

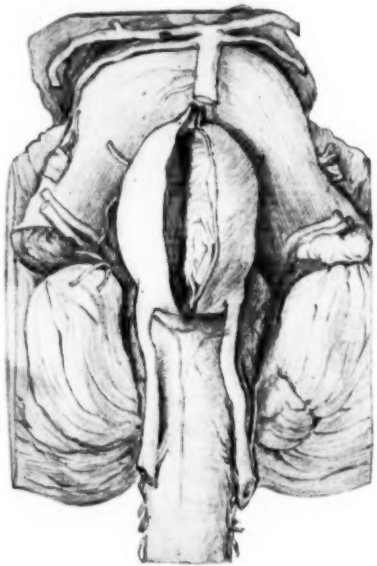
B

C

PLATE II.

PLATE II.

A



B

PLATE III.

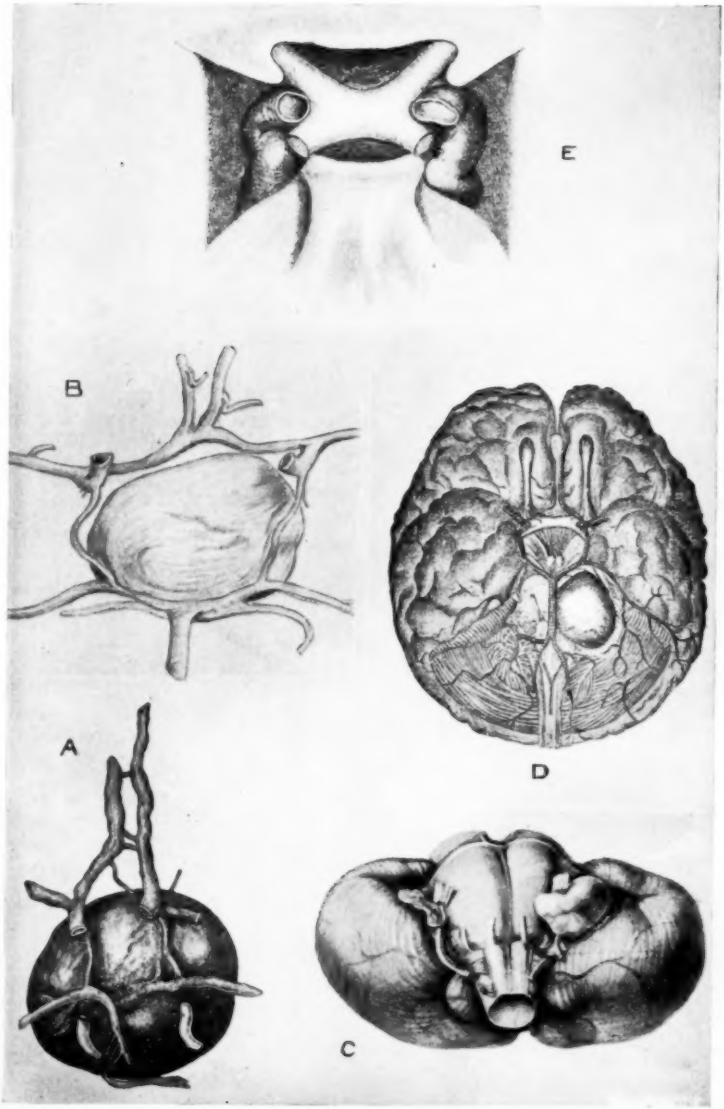


PLATE IV.

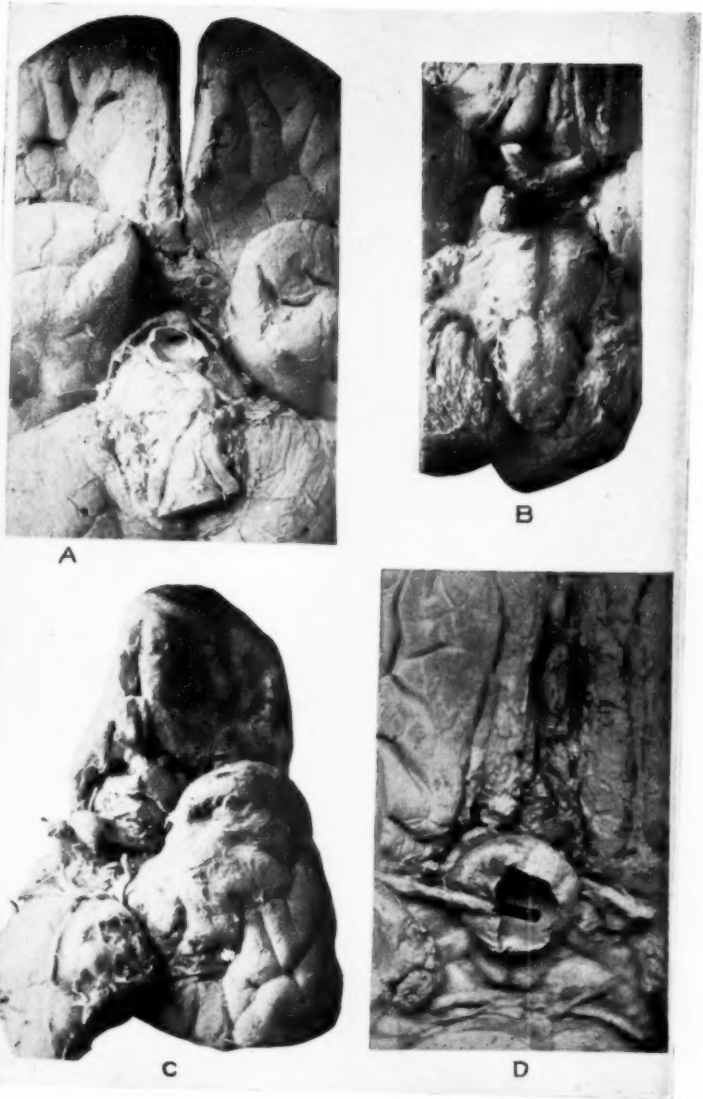
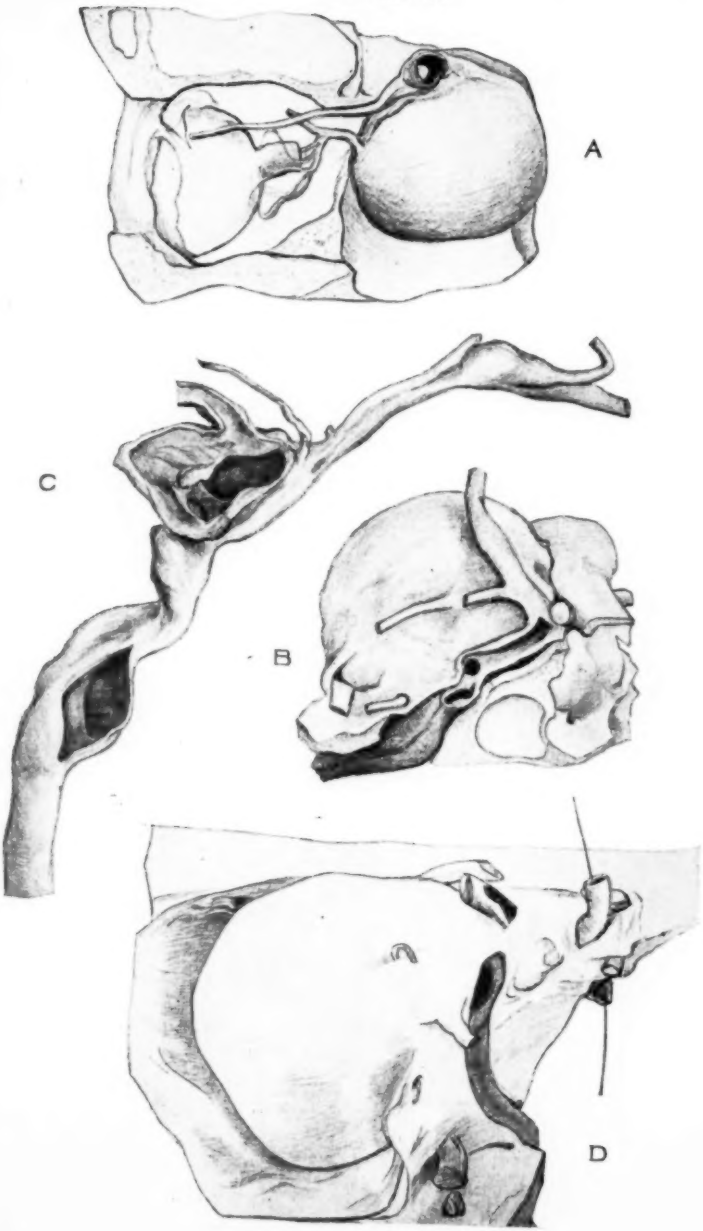


PLATE V.



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# TWO CASES OF DISSEMINATED SCLEROSIS, WITH AUTOPSY.

BY J. A. ORMEROD, M.D.

THIS paper gives an account of two cases of disseminated sclerosis, in which the diagnosis was established *post mortem*. The cases in which this diagnosis is made clinically, and no doubt correctly made, are nowadays very numerous, but completed cases seem to be uncommon. Partly for this reason, I venture to publish these two cases; partly, too, because the clinical symptoms appear to me interesting, and very different in the two patients, though both had the same disease.

In the first case, the disease was not diagnosed during life. There are many cases of disseminated sclerosis admitted to Queen Square Hospital, concerning some of which there can be little doubt as to the correctness of the diagnosis, while others of them present symptoms which are suggestive rather than convincing as to their nature. The bulk of these patients leave the hospital sometimes considerably improved, sometimes as incurable paralytics, and an anatomical proof or disproof of the diagnosis is never obtained. But in the present case this order of things was reversed. Disseminated sclerosis was not suspected during life. Indeed, there were no facts to suggest it. The patient's age, 54, was against it. The symptoms began in a very acute way. Pain was a prominent feature. The leading symptom at the time of admission, *i.e.*, intense flexor spasm of the legs, may certainly be seen in long-standing cases of disseminated sclerosis; but this patient had only been ten months ill. Thus the *post-mortem* discoveries were in every way a surprise. Nor did the localisation of the disease *post mortem* adequately explain the clinical characters of the

case; for in the cord most of the patches were found in the cervical region, yet there was no affection of the upper limbs, at least till a week before death. The dorsal and lumbar regions were less affected, and the descending degeneration of the pyramidal tracts was slight. Yet there was intense contraction of the legs, which had existed in one form or another from the very commencement, and incontinence of urine.

I fear that the rapid termination of the case must be connected in some way with the operation for straightening the legs. This procedure, though necessary and in no way severe, involving indeed no loss of blood, appears to have been too much for the resistance of an enfeebled nervous system.

The second case is remarkable for the hysterical phenomena which occurred in connection with it, and for a peculiar condition of the skin. The association of hysterical symptoms with disseminated sclerosis is, of course, no new observation; but I have seldom seen the diseases so closely interwoven as in this case. The patient (when first seen by me) had an extensive hemianæsthesia, which could be shown by Janet's test to be psychical in character; she had a rigid condition of the fingers, which yielded to forcible passive movement, though at the cost of some pain; she had an inability to walk which must have been largely "functional"; for though she had been bedridden for many months, she was able, after about five weeks' treatment, to get about the ward; and, indeed, had it not been for the presence of Babinski's sign and of optic atrophy, I think the diagnosis of hysteria, pure and simple, might have been justifiable. Yet the *post mortem* showed that she had, as was thought during life, disseminated sclerosis.

The rash was a very striking object when I first saw her, but the principal period of efflorescence was then over. She stated that it began as raised papules, which turned into "blisters," and this seems credible, for one bulla was actually observed. But most of the spots which developed in hospital began either as a small papule, which slowly increased in size and eventually formed a small sore; or as

a raised papule capped with a small excoriation, or as an abrasion or excoriation, generally of an elongated shape. The old healed places showed as oval patches of superficial atrophy, whose white colour was shown up by an edging of dark pigment. The rash only occurred on anæsthetic skin. Various views might be entertained as to the origin of it, *e.g.* :—

(1) That it was the expression of some organic nervous lesion. But no lesion was found *post mortem* with which it could be connected.

(2) That it was like the anæsthesia, hysterical. The existence of hysterical rashes is asserted by some authors, and as flatly denied by others. I myself have never seen anything like the present rash in cases of hysteria.

(3) That it was manufactured by the patient. There was much to be said for this view, but even after prolonged observation I could not prove it.

(4) That the anæsthetic skin had undergone some sort of nutritional change, in virtue of which it reacted in this peculiar way to slight stimuli which would have no effect on a healthy skin. As against this view, it should be noted that there was no rash on the (anæsthetic) right arm.

### Case I.

*Onset sudden, ten months before admission. Extensive spasms of legs, with diarrhœa, repeated on four successive days. Further cramps in legs five weeks later, with pain, finally assuming the form of flexor spasms, lasting for some months. Gradual development of flexor contracture of the lower limbs.*

*Painful tinglings in legs six weeks after onset; incontinence of urine, with anæsthesia of the bladder. Rigidity of abdominal muscles two months before admission. Pain in back and lower limbs.*

*On admission, lower limbs firmly flexed at hip and knee; much tenderness to touch or manipulation; incontinence of urine. Legs straightened under chloroform two and a half months after admission; patient died of exhaustion some nine days later.*

*Post mortem.—Viscera normal; plaques of disseminated sclerosis in spinal cord, principally in the cervical region, in the pons and medulla, right optic thalamus, and right optic tract.*

*Case I.*—(The notes of the case were taken by Dr. S. A. K. Wilson.) Charlotte L., aged 54; widow during last twenty-six years. Admitted to Queen Square Hospital under the care of Dr. Ormerod on July 21, 1905. Her father lived to 86, and her mother to 77; she had six brothers and sisters; one of them died of consumption, the rest were healthy. She had three children, two of whom died of consumption (there appears to be consumption in her husband's family).

She had fair health in early life. Eighteen years ago she had rheumatic fever, and scarlet fever at the same time; since this her heart has been "weak." She had bad influenza three times, the last attack eleven years ago. There has been no return of rheumatic fever, but she had "rheumatism in the muscles of the back" six years ago.

It appears also that four years ago she had what she calls a stroke on the right side of the body; this lasted for twenty-four hours. Possibly this may have been a premonitory symptom of her present illness.

The present illness began quite suddenly on the afternoon of September 22, 1904. She was in her usual health, and was sitting resting, when, without any warning symptom, her legs became straight and stiff, with the toes spread out. At the same time she had violent diarrhœa. The legs remained in this condition for about two hours, after which time she could again bend them voluntarily. The diarrhœa lasted rather longer. There was then no pain in the back, no tingling or numbness, and no trouble with micturition. She knew of no cause for the attack, which took her completely by surprise.

The next morning, from 7 a.m. till 12 noon, she again had an attack of diarrhœa and extensor spasm of the legs; and on the two following days, during the same time, the attacks occurred again. After this it would appear that the legs, especially the right, began very slowly and gradually to draw up, the knee slowly approaching the trunk.

Six weeks after the first attack (in the beginning of November 1904) she began to have difficulty in holding her urine, and this incontinence has increased. In January 1905 she lost all sensation of the act of micturition, and cannot now tell whether the bladder is emptying itself or not, except by the feeling of warmth on the thighs.

About the same time, *i.e.*, about six weeks after the onset, a painful tingling sensation began in the legs "like a wound-up clock running down." It always seemed to run from the knees

to the toes. It usually came on in the evening and lasted several hours. This lasted for some months, but stopped about February 1905. In November 1904 bedsores began to appear, which lasted through the winter and spring.

To return to the muscular spasms. After the first three days these practically disappeared for five weeks. They then recurred in rather a different form, viz., as violent and painful cramps in which the legs went out straight and then drew up again quickly; these cramps were associated with severe tingling pains in all the muscles from the hips downward. About Christmas, 1904, these cramps were replaced by flexor spasms, *i.e.*, she had attacks of pain over the hips and down the legs, during which the legs were drawn up spasmodically without any previous extension.

All this time the legs had been slowly contracting, and by Good Friday, 1905, the contraction had reached its worst, the right knee being drawn up, she says, under the left axilla, and the left similarly contracted. An operation for straightening them was undertaken, but soon afterwards they began to draw up again till they came to their present position. She cannot move them voluntarily, not even the toes. There has never been any loss of sensation; indeed they have been always, and are now, very sensitive.

She had had, almost from the onset, a tight feeling round the waist, but about two months ago the trunk muscles became more obviously affected, in that the patient had occasional pain across the belly, which became hard like a board. This condition is permanent, not paroxysmal. Constipation has taken the place of the initial diarrhœa.

During the last month she has suffered more definitely from pain in the back; this is severe, usually worst when the pains in the legs come on; it then shoots up into the spine, and she feels as if the whole of the spine were being forcibly doubled backwards.

She does not think the legs are wasted; she says that they were very swollen at first, but that this soon disappeared. There has been no affection whatever of the upper limbs.

CONDITION ON ADMISSION.

Patient is somewhat emaciated; she lies on her back with her legs drawn up and crossed, the right underneath the left. The legs are œdematous. There is an old bedsore on the sacrum. She has pain when the legs are touched or moved, but not otherwise. The abdominal muscles are rigid. Pulse 80; the heart is

somewhat enlarged, the apex being an inch outside and below the nipple, but there is no murmur. There is no evidence of lung disease. The urine contains no albumen.

*Examination of Nervous System.*

The cerebral functions are natural, though the pain makes her emotional. There is no affection of the special senses, except that she is deaf, especially with the right ear. In the district of the cranial nerves there is no definite abnormality except for a slight facial asymmetry, a slight and occasional ptosis of the right eyelid, and a little difference in the movements of the eyelids when she winks. The optic discs are both rather pale in the temporal half.

*Motor Functions.*—She cannot stand. The muscles generally and those of the legs in particular are small and flabby; the wasting in the legs is partially concealed by œdema. The legs are contracted; the right is flexed at the hip and knee and strongly adducted, so that it lies right across to the left side; over it is the left leg, similarly flexed at the hip and knee, but not adducted. Any attempt to overcome this contraction causes intense pain. The rigidity seems to be more in the joints than in the muscles, except as regards the hamstrings, the tendons of which one may feel to become taut. The gastrocnemii are peculiarly flabby and toneless. The glutei seem flabby, though not obviously wasted; their condition is more or less masked by œdema.

There are no fibrillary twitchings, no tremors, no reflex spasms, nor involuntary movements. Voluntary movements are normal in the head, neck, and upper limbs; in attempting to sit up the rigid abdominal muscles become somewhat harder than before; the erector spinæ cannot be tested. In the lower limbs the only voluntary movements obtainable are slight flexion and extension at the ankles, better on the right than on the left side. The attempt to move sometimes produces on the right side an incomplete spontaneous ankle-clonus.

*Sensory Functions.*—There is intense hyperæsthesia to touch and to prick in the legs generally, and particularly over the ankles and the soles of the feet. Stroking the sole of the foot to produce a plantar reflex seems to produce intense pain. Similarly, pressing the feet to estimate the œdema produces much pain. Any attempt at passive movement of the legs, particularly at the hips or knees, causes intense pain. There is pain in the back, about the level of the sixth dorsal area, and again very low down in the

small of the back, and also in the abdomen, coming round from the small of the back.

No definite anæsthesia can be made out, but she seems to feel a pin-prick more acutely over the abdomen than over the pelvis or thorax. There is no loss of the sense of position or of passive movement in the legs or in the toes. The sense of pain when the muscles are pressed is much increased.

*Reflexes.*—In the upper limbs the deep reflexes are all brisk and equal. The knee-jerks cannot be obtained on either side, probably owing to the position and contraction of the legs. The ankle-jerk seems to be present on both sides, though slight.

No abdominal or epigastric reflex can be obtained on either side.

*Plantar reflexes.*—Not obtained with certainty; only occasional toe movements follow the stimulation. (Later, however, plantar reflexes of extensor type were obtained.)

There is incontinence of urine as described above.

*Vaso-motor and trophic functions.*—The legs are œdematous, especially about the ankles. The skin is very thick, coarse, and dry, scaling off in flakes over the toes and feet. The nails are dry and cracking. There is an old bedsore over the sacrum.

October, 1905.—The patient is no better, if anything worse; the left leg is more contracted; she complains constantly of pain. The position of the legs gives rise to much difficulty in the nursing.

Consequently it was decided to straighten them under chloroform. This was done on October 7, at 3.30 p.m.; many adhesions were broken down, the limbs were made much straighter, and the knees separated. She recovered fairly well, but remained somewhat drowsy, and got worse towards the evening; the temperature rose to 100° F., the respirations became quick and shallow, and her colour rather blue.

October 8.—Temperature 100°; pulse and respiration are hurried, but examination of heart and lungs shows nothing abnormal. Drowsy and semi-conscious; right eyelid droops more than usual; swallows with difficulty. The arms are very rigid and resistant to passive movements.

October 15.—During the week the patient has been steadily failing. There has been continuous slight fever (99·5—101°), drowsiness, occasional cough; physical signs of hypostatic congestion at the basis of the lungs. The condition of the legs has remained unchanged; the plantar reflexes are of the extensor type on both sides. There appears to be more definite weakness on the right side of the face.

October 16 (6 a.m.).—The temperature rose to 105·8° F., and the patient died quietly soon afterwards.

The *post mortem* was made seven hours after death by Dr. Gordon Holmes, who has furnished me with the following report:—

AUTOPSY.

*Body* well nourished, *rigor mortis* present in all limbs, knees contracted and cannot be straightened out. Both feet œdematous.

*Visceral organs* normal, except that the lower lobes of both lungs are congested.

*Vertebral column and skull*, normal.

*Spinal cord.*—Roots and meninges appear normal; cord of fair size, and no discoloration visible on surface. On drawing finger down cord a sensation of ridges is obtained, as though the consistence of the tissues were irregular. On section through it there seems to be irregular patchy disease in the cervical and dorsal segments.

*Brain.*—Vessels and meninges appear normal. No wasting nor other evident disease of the cortex; no patches of sclerosis visible in it. On cutting sections of the brain, the only patch found is in the posterior part of the right optic thalamus. Small patches of discoloration are visible on the ventral surface of the pons. (When further sections of the brain had been made after hardening, several small foci of disease were seen in the pons, and one in the mid-brain in the neighbourhood of the right external geniculate body.)

*Microscopical Examination.*

*Cord* (stained by Weigert-Pal method).—The cervical segments contain the chief evidence of disease. Here there are numerous sclerotic patches in which no myelinated fibres stain; they have, as a rule, a sharp border, though in some regions the sclerosis is more diffuse, and some medullated fibres may persist in the area which appears diseased. The patches do not seem to have any special site of predilection; grey and white matter are equally affected.

In the dorsal and lumbar segments there are fewer definite patches such as are typical of the disease; but foci of diffuse sclerosis, in which some myelinated fibres persist, may be seen in many segments.

There is in addition slight descending degeneration, at least of the myeline sheaths, of both pyramidal tracts; but that of the one side (? right) is considerably more affected than the other.

No definite ascending degeneration is visible in the posterior columns, nor in the spino-cerebellar tracts.

*Brain-stem.* — A large patch of sclerosis is visible just at the decussation of the pyramids, but no others were discovered in any of the levels of the medulla which were cut.

In the pons is a patch lying in the floor of the fourth ventricle, and at another level a large focus just ventral to the seventh nucleus.

In the mid-brain are a few small foci, situated in the ventro-lateral periphery of the section, at about the level of the posterior quadrigeminal bodies, and two patches are seen in the right thalamus, the larger of which, measuring about $\frac{1}{2}$ cm., lies in the pulvinar.

In sections through the chiasma it is found that one optic nerve had lost all its myelinated sheaths for a considerable distance, and there is some diminution in the myelinated sheaths of the other. The eyes were not examined.

Bielschowski's stain show that the greater number at least of the axis cylinders in the sclerotic patches are normal.

Sections prepared by Weigert's method for demonstrating the neuroglia were not very satisfactory, but they show a dense sclerosis of neuroglia in the foci of disease.

No evidence of vascular disease in the central nervous system can be found.

### Case 2.

*Woman of middle age, who had had many illnesses. Lymph-angitis with abscesses of right upper limb; splint applied; stiffness of elbow and fingers followed, and paræsthesia in fingers, spreading to right side of trunk. Afterwards abscess right thigh, weakness right lower limb; peculiar rash right thigh and right side of body.*

*On admission to St. Bartholomew's* (*January* 13, 1905) *three or four years after onset she was completely bedridden. Right upper limb was rigid, and particularly the fingers, where there was great pain on passive movement; right hip rigid, but less so than upper limb; wasting of right limbs, especially the upper limb; right hemianæsthesia, and a rash of peculiar appearance and distribution. Plantar reflexes extensor; optic atrophy* (*observed later*). *During next few weeks much improvement in motor condition of right limbs; slight spread of the eruption.*

*Transferred to Queen Square four and a half months later* (*May* 29). *Still extreme rigidity of right fingers; anæsthesia has*

*spread upwards to right clavicles; rash now stationary. No further change for the next three or four months, then (September-October.* 1905) *spread of symptoms to left side, viz., anæsthesia left as well as right from neck downwards, weakness of left limbs, jumping movements, and disagreeable sensations in left limbs; further slight spread of rash; general debility and depression.*

*She left Queen Square at Christmas,* 1905, *and died of abdominal cancer in November,* 1906, *i.e., some five or six years after the onset.*

*Examination of the nervous system showed the presence of disseminated sclerosis.*

*Case* 2.—This patient was an inmate of Tonbridge Infirmary, under the care of Dr. Crawford, before she came under my observation. I must thank Dr. Crawford for sending her to me, and for much valuable information and help. I must also thank Dr. W. T. Storrs for drawing my attention to the case in the first instance.

Alice L., aged 38, single, was admitted to St. Bartholomew's Hospital, January 13, 1905.

In her family history there is nothing that needs mention.

She herself has never been robust. As a child she had "croup," frequent sore throats, and bronchitis. At the age of 13 she had rheumatic fever; at 17, muscular rheumatism in the legs, which laid her up for three or four months; at 19, severe diphtheria; at 22, "congestion of the lungs"; at 31, typhoid fever; at 33, an illness which began with an ulcerated throat, and which led to cough and short breath, with expectoration of blood and offensive pus. She was ill for three months.

During convalescence from this, *i.e.*, between three and four years ago, her present illness began, and in the following way. She scratched her right thumb on a brass bracket, lymphangitis followed, and an axillary abscess which discharged for a month or more; then an abscess appeared at the elbow, lasting several weeks. The elbow was put on a splint; it has remained stiff ever since. The fingers of that hand also became stiff; the limb became wasted and weak. Pricking and tingling sensations began in the fingers of the right hand (not in the thumb apparently) about a month after the accident, associated with a feeling of numbness, and this numbness spread up to the shoulder and on to the right side of the trunk. It appears from statements made to Dr. Wilson at Queen Square that similar sensory pheno-

mena, viz., tingling and numbness, began in the right foot and toes, particularly the great toe, about the same time as in the hand, and extended up to the hip, though the numbness was not always present; the limb felt weak to her, and the ground under the foot "felt soft and funny."

All active symptoms, however, had subsided a year after the injury, but the arm had settled down into its present state of rigidity and uselessness. She remained in this state about nine months.

Then an abscess formed in the front of the right thigh which laid her up again, and while this was still discharging the leg got gradually weaker, and an affection of the skin began. A series of spots, about half a dozen in number, appeared in the neighbourhood of the abscess—raised red papules, about the size of a sixpence, which in twenty-four hours turned into blisters, and finally formed sores which took a long time to heal. This was in February, 1904. In May a fresh crop appeared, and they continued to come out during the rest of the year, spreading down the right thigh and leg, and upwards over the right half of the trunk. Over seventy were counted.

The outbreak of a fresh spot was preceded by local pricking and tingling; they were painful, but did not itch.

She has been in Tonbridge Infirmary more than a year. Dr. Crawford writes me word that, having suspicions as to the origin of the rash, he directed the nurses to watch her carefully, but they never detected her manufacturing it. She had also severe general pains which kept her awake, and for this she had been given, before her admission to the infirmary, morphia subcutaneously, which relieved her insomnia, but gave her severe headache in the morning. Hypodermics of plain water were substituted in the infirmary, and produced exactly the same result. She was given a long course of arsenic, which may have caused the peculiar pigmentation round the rash.

### Condition on Admission to St. Bartholomew's (*January* 13, 1905).

(The notes are mainly abridged from those taken by Dr. G. B. D. Adams, then house physician.)

Patient is a woman of rather spare build. Complains of pain in the right hip. She lies quietly in bed with the right arm close to the side, the right elbow (which is stiff) semiflexed, and the forearm laid across the body. The temperature, pulse, and respiration are normal. There is some lateral curvature of the spine, convexity to the left, in the dorso-lumbar region, and some

slight deformity of the chest owing to prominence of the third rib on both sides. Examination of the chest and abdomen shows nothing abnormal. The teeth are bad; her hearing is good; she has bad eyesight, for which she wears spectacles. The pupils are normal, the ocular movements are normal, there is no nystagmus. An examination of the fundus oculi was unfortunately not made, or at least not recorded.

The facial movements are the same on both sides; there is no loss of sensation in the face.

*Condition of the Limbs.*—The left arm and left leg are natural in appearance, and their movements are natural; but on the right side the upper arm is slightly wasted ($7\frac{1}{4}$ ins. circumference as compared with $8\frac{1}{4}$ ins. on the left); the muscles of the forearm and hand, especially the interossei, are markedly wasted. The movements at the shoulder-joint are good, those at the elbow limited; flexion is possible to an angle of about 45°, extension to an angle of 120°, but the joint is not definitely deformed or thickened. The movements of pronation and supination are free, those at the wrist-joint free but weak. The hand is useless, the metacarpo-phalangeal joints are slightly flexed, and there is a corresponding over-extension of the proximal interphalangeal joints; passive flexion of the fingers is possible, but there is much resistance to it, and it gives her great pain. She cannot voluntarily flex the fingers nor separate them.

The right lower limb looks shortened at first sight, but this is apparent only, and due to tilting of the pelvis and adduction of the limb; measurement shows it to be of the same length as the left. The limb is adducted and very slightly inverted, but not flexed. Tapping over the great trochanter or on the sole of the foot causes pain in the hip-joint. The movement of the right hip-joint is very limited; passive flexion is possible only through an angle of 25°, and causes pain. The muscles of the thigh are wasted (circumference $13\frac{1}{4}$ ins. as compared with 14 ins. on the left side); and those of the calf are also rather smaller than the left.

There is no affection of micturition or defæcation. The knee-jerk is more marked on the left side than on the right; there is no ankle-clonus on either side. The plantar reflex is difficult to obtain on the right side, on the left side it is distinctly extensor in type. An extensor plantar reflex was subsequently obtained on the right side also.

There is anæsthesia to touch, prick, and thermal stimuli over the right arm, the right leg, and the right half of the lower part of the trunk. In some places, however, the patient still feels a prick (see fig. 1 made out by Mr. J. G. Watkins, clinical clerk).

The rash, which formed a striking feature of the case, was distributed over the lower half of the right side of the trunk, the right thigh, and part of the right leg. Part of the buttock adjacent to the fundament had no rash upon it. (See fig. 2 by Mr. Watkins, and the photograph.) Thus, though the whole of the anæsthetic area was not affected (the arm, for instance, being free), yet there was no rash on such parts of the skin as retained sensation. On the trunk it was most profuse from the groin to the level

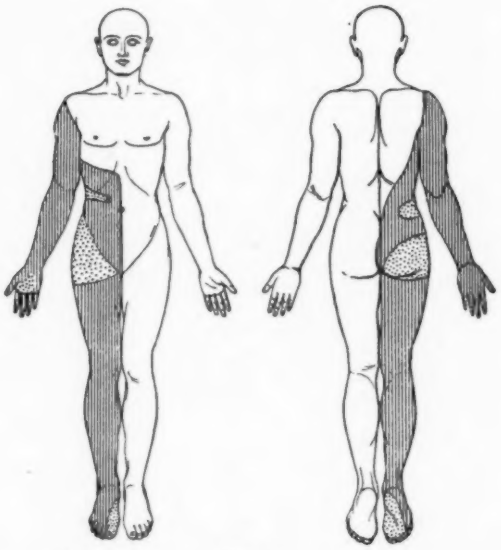

FIG. 1.

Distribution of anæsthesia, January 14, 1905. Anæsthesia to touch, prick, and temperature, over dark area; over dotted areas, a prick is still felt. (Mr. J. G. Watkins.)

of the eighth rib in front, and from the buttock up to the lower angle of the scapula behind; it stopped abruptly at the middle line, both in front and behind, except where some pigment spread across the sacrum to the inner aspect of the left buttock. There was rash all over the thigh, and a few separate patches on the outer side of the calf; none on the shin.

It consisted of spots and patches which were for the most part discrete. The most recent forms appeared to be red, slightly

raised papules, about half an inch in diameter, with a small abrasion at their apex. Other forms were shallow ulcers or abrasions, where the cuticle had been lost, leaving raw red patches of an oval shape for the most part, and from one to two inches long. These oval lesions were placed vertically (in the long axis of the limb) on the thigh, in the groin and neighbourhood obliquely (parallel with Poupart's ligament); on the trunk they were irregularly disposed. Each was surrounded by a zone of brown pigmentation. There were also older lesions, consisting of white

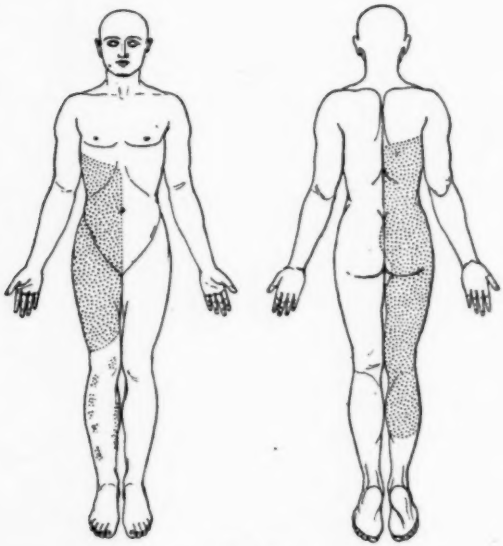

FIG. 2.

Distribution of the rash, indicated by dots. January 14, 1905. (Mr. J. G. Watkins.)

scars, oval or irregular-shaped, each surrounded by a zone of pigmentation. On the front of the thigh the scarring and pigmentation were confluent. There was no indication of the site of the abscess, which was said to have occurred in the front of the thigh.

It will thus be seen that the case presented peculiar symptoms, difficult to interpret; a cursory review of them suggested some such diagnosis as the following:—

That owing to some infection the elbow-joint or the bone of

the upper limb had become diseased, and that the nerve trunks had become involved; hence paralysis with atrophy of the hand muscles, and possibly distortion of the fingers.

That the hip-joint was similarly diseased.

That the unilateral anæsthesia and unilateral rash might be due to some obscure (infective ?) disease of the spinal nerve-roots on the right side.

But these suppositions were disproved by further investigation of the facts. For in the first place, Mr. Lockwood, who kindly examined the joints for me, said that they were not diseased. Secondly, Dr. Lewis Jones reported that the electrical reactions of the wasted muscles were normal; thus the existence of a neuritis, or of disease of the anterior cornual cells, was negatived. Thirdly, the anæsthesia was proved to be psychical in the following way:—

After ensuring that she could not see what was being done, the patient was first touched several times on the sound parts, and told to reply "yes" each time she was touched. She did so correctly. She was next touched on the anæsthetic part, and told to reply "no" each time she was not touched. She did so, equally correctly.

After a few trials, this test (as usual) began to fail, so it was varied. Her attention was drawn to the paralysis of her right hand rather than to the anæsthesia, and she was told first to touch with her right forefinger any part that I touched. I touched a sentient part of her body, and she laboriously touched it with her right forefinger. I then told her to do the same with her left hand, taking care to remind her that this was her good hand. I touched an anæsthetic part, and she followed suit quite readily. This test also began to fail after a time, but in a curious way. When told, as before, to follow with her left hand, she would lift it up to do so and then drop it as if she could not carry out the command, but betraying all the same that she had felt the touch. And yet, all the time, the anæsthetic parts, when tested in the ordinary way, were anæsthetic to stimuli of the severest kind.

I concluded from this examination that the anæsthesia was hysterical, and thought it probable that the paralysis was also largely hysterical. The rash remained a puzzle, but it seemed to me not impossible that the patient had produced it herself. The leg was covered with boric acid ointment and bandaged, and a careful watch was kept for the appearance of any new spot, and the circumstances under which it might arise. Although some of the notes read rather like conviction of a fraud, I think I may say the verdict was "not proven" one way or the other.

I give the notes verbatim as regards the rash.

January 20.—A few new spots have occurred, especially lower down the calf of the right leg.

January 21.—A new blister appeared yesterday, containing clear fluid. It collapsed this morning. There is now a reddened oval area 1 inch long, without pigmentation round it.

January 31.—Two or three new spots have appeared in the right axilla and thigh.

February 7.—Two or three more spots on the back of the leg and in the axilla.

February 21.—One or two new spots.

March 14.—One or two new spots, having the character of abrasions.

March 21.—Scratched left hand with a pin, rubbed it with the fingers of the right hand, and made a sore place, discharging pus.

One or two new spots.

March 28.—Two or three (new) small shallow ulcers on the inner side of the right ankle.

April 4.—One new ulcer since last week on the inner side of the right ankle.

April 11.—Three new lesions transversely placed across right instep, produced by friction with left heel (observed).

May 16.—Two new lesions in groin, and two just below the knee.

The excoriations which had been present on admission were healed by March 8. Of the new spots which developed, some were in points accessible to the patient, some under the bandage. Most of them took the form of red raised patches with an abrasion on their summit, though a definite bulla was once observed (January 21).

The patient was shown to the Dermatological Society of London on March 8. The general view taken by the Society was that the rash was manufactured; but one member, whose opinion should be of great weight, dissented.

The patient was treated with passive movements of the limbs, and the electrical wire brush. The anæsthesia did not clear up, but the motor condition improved remarkably. Thus, whereas she had been bedridden for many months before admission, by February 21 she could walk with very slight assistance, and by March 7 she was walking alone, though she still kept the right leg stiff as she walked. The elbow also soon became flexible, but very little impression was made on the condition of the fingers; they re-

mained in their peculiar position. Passive flexion of these was possible, but caused her intense pain.

A note taken by me on March 7 says that the anæsthesia is still present; the knee-jerk on the left side is easily obtained, on the right it is obtained with reinforcement; there is no ankle-clonus; the abdominal reflex is not obtained; the plantar reflexes are extensor, the left typically so.

It was recognised, in the presence of this distinct Babinski's sign, that the disease was not wholly hysterical.

She was transferred to Queen Square Hospital on May 29, 1905.

The notes in Queen Square Hospital were taken by Dr. S. A. K. Wilson, who took a great interest in the case; I have abbreviated them for the purpose of this paper.

The history corresponded in the main with that just given; one additional point is that the patient stated that for the last seven years she had had to hurry somewhat to pass urine, *i.e.*, had had some degree of "precipitate micturition." She herself acknowledged the improvement in the movements of the lower limb and of the elbow that had taken place while she was at St. Bartholomew's, but in the hand she said (correctly enough) there had been little change.

CONDITION ON ADMISSION TO QUEEN SQUARE (*May* 29, 1905).

*In the district of the cranial nerves* there is no abnormality, with the one very important exception, that she has double *optic atrophy*. Mr. Marcus Gunn's note on this point (June 5) says: "Both discs are decidedly pale and atrophic in appearance; the right is the better looking of the two, there being still a pink tinge over the inner half. Vessels of good size, lamina cribrosa well seen. Whole appearance is that of a simple optic atrophy. Eyes nearly emmetropic."

The colour vision (central) is normal on both sides; the field (for white) is reduced to a very small central area.

*Motor Functions.*—The general muscular development is fair, with the exceptions to be presently noted. There is no kind of involuntary movement.

Of the upper limbs, the left is normal; in the right there is slight general wasting, especially below the elbow; passive movements at the shoulder give rise to some resistance and pain; voluntary movements are also limited here, as if from stiffness of the shoulder-joint; movements at the elbow and wrist are good in range, though somewhat weak; but the fingers are rigid and

fixed in a peculiar position, viz., the interphalangeal joints are fully extended and very rigid, but the terminal phalanges are flexed on the others (particularly in the first, second, and fourth fingers). She cannot close her fist at all; the attempt results in an interosseal position. Any attempt at passive flexion of the rigid joints gives rise to intense pain. The thumb, however, is not affected, and she can move it well.

As to the lower limbs, the left is natural; there is slight wasting and flabbiness of muscles in the right, at the right hip there is some pain and resistance upon passive movement, and the voluntary movements are weak. Similar resistance is noted at the knee, and there is some grating of the patella. Voluntary movements of the ankle are fair in range, but feeble in power; movements of the toes are fair.

There is no incoördination, so far as can be made out in the presence of feeble and defective movement.

*Sensory Functions.*—Subjectively, there is pain in the right shoulder, knee, and hip upon passive movement, and very great pain when an attempt at passive flexion of the right fingers is made. There is numbness on the right half of the body, from the neck downwards.

Objectively, it will be seen by the chart (fig. 3), as compared with those made in St. Bartholomew's, that the anæsthesia has spread up to the level of the clavicle. From this point downwards the whole right half of the body is completely anæsthetic to touches and to pricks, except for some parts indicated by dots on the chart, viz., the upper edge of the anæsthetic area, the thumb, especially the tip of the thumb, the hypogastrium and region of the anus, and parts of the sole. Here a prick is felt as a touch. Janet's test, however, can no longer be obtained. The perception of heat and cold and the perception of vibration (pallæsthesia) are also lost over the right half of the body from the clavicle downwards, again excepting the thumb. The sense of weight as tested by balls, the muscle pain sense, and the sense of position are all absent in the right limbs. Thus she cannot tell when the limbs (right) are being moved passively, excepting the right thumb: she cannot imitate with her left arm the position into which her right arm has been put; indeed, she does not know where her right arm is till she opens her eyes and looks at it. As to stereognosis, if the patient gets an object between the right thumb and forefinger, she makes a fair attempt at guessing what it is, but in any other part of the hand there is complete astereognosis. It may be, however, that she is hindered from recognising objects by the rigid state of the fingers.

On the left side of the body, and in the left limbs, all forms of sensation are normal. There is no loss of visceral sensation; for instance, she knows when the bowels move and when her urine passes.

*Reflexes.*—Tendon reflexes are normal in upper and lower limbs (excepting that a contralateral adductor reflex is present, on testing the knee-jerks on both sides). There is no ankle-clonus.

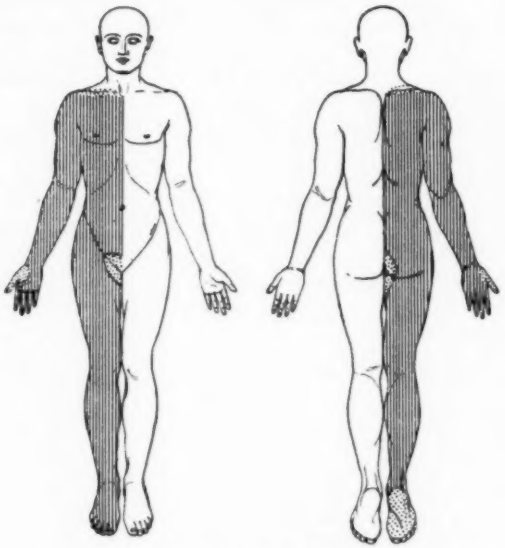

FIG. 3.

(Dr. S. A. K. Wilson.) May 29, 1905. Over dark areas, complete anæsthesia to touch, prick, temperature, vibration. Over dotted areas prick is recognised as a touch. At tip of thumb it is recognised as a prick. Over thumb, too, sense of temperature and of vibration is preserved.

The abdominal and epigastric are present on both sides; the plantar are of the extensor type on both sides, but the left is more marked, or, at least, more easily obtained than the right.

The organic reflexes are normal, except it be for the precipitancy of micturition mentioned above.

*Gait.*—The patient advances rather slowly, dragging the right foot. The chief step in advance is always taken with the left foot; the right is then slowly brought forward and shuffled along

the ground to a position very little in advance of the left. On the right foot the chief weight is thrown on the heel, the toes are sometimes off the ground. There is no ataxia of gait, and no Romberg's sign.

As to the condition of the skin, it is unnecessary to describe the rash in detail again. There are now only two or three of the spots which have not quite healed; and these healed places present the appearance of pale, non-depressed scars, mostly oval in

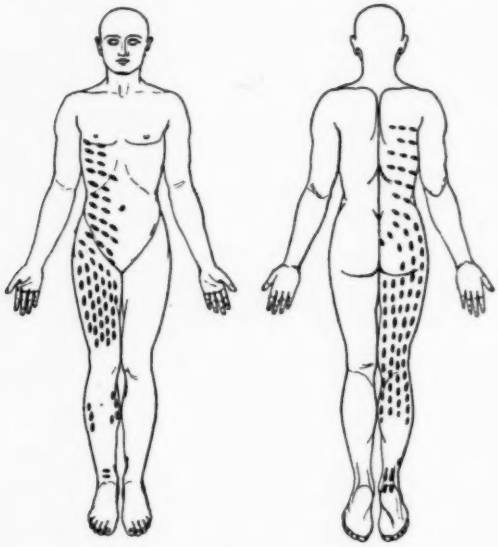

FIG. 4.

(Dr. S. A. K. Wilson.) May 29, 1905. Distribution of the rash, and direction of the long axes of the oval spots.

shape (the long axis having the directions described in the former note), surrounded by a deep zone of pigment, and closely packed.

*Electrical Examination.*—The muscles on the right side require a stronger faradic current to produce reaction than do those on the left; to galvanism there is no reaction of degeneration. Over the anæsthetic area she bears a very strong current, apparently without feeling anything—a current strong enough to produce firm tetanus of the muscles—and which on the left side is quite unbearable.

Later (July 15) a lumbar puncture was made; 15 cc. of fluid was withdrawn under considerable tension; the fluid was clear, and, when examined histologically, proved to be quite normal.

During the summer no particular changes were noted in the patient's condition; her general condition remained good, her weight increased by a few pounds (May 29, 7 st. 9 lbs.; October 1, 8st 1 lb.); her temperature was never above 99°; but in the autumn her nervous state, and particularly the sensory condition, began to get worse. Thus a note of October 15 says:—

October 15, 1905. For the last few weeks the patient has had occasional sensations which she tries to describe as follows: "Sometimes I feel as if I weren't anywhere. Yesterday I felt this in the day room; I had to think whether I was there or somewhere else. I feel constantly as if there were no right half to my body, as if it did not belong to the other half."

About six weeks ago she noticed that the left hand and arm were becoming numb. This began with a sensation of pins and needles in the ulnar fingers, spreading up to the elbow. About a week ago it had reached the shoulder. She has felt increasing weakness and awkwardness in the left arm and has been dropping things. Her writing, which used to be wonderfully good with the left hand, is distinctly worse. The sensations in the left arm are distressing her very much because the paralysis of the right arm began, she says, exactly in the same way. Within the last few days this numbness of the left arm has spread and become much more intense. "I feel nothing on that side now; when I take anything in my left hand I must look at it, otherwise I do not know that I have it. I do not know whether my left hand is open or closed unless I look at it."

Within the last week also a sort of irregular spasm has been noticed once or twice in the left arm and leg, consisting in occasional twitches of various muscles and muscular groups. On occasions it is much worse, she says: "Sometimes my leg nearly jumps off."

A careful re-examination of her general state was made (October 15) with the following results:—

There is no particular change in her general appearance. She appears anxious to improve; she does not know why she is getting worse. She has headaches and sleeps badly. She has a cough, obviously neurotic, which increases when attention is paid to it.

*Cranial Nerves.*—There is no change in the optic discs; there is a slight tendency to nystagmus upon extreme lateral deviations in either direction.

*Motor Functions.*—The weakness of the right side increases, the fingers are perhaps less stiff, but there is no return of power and they are excessively painful when passively flexed, in spite of the general and complete anæsthesia. There is, in addition, weakness of the left arm; there is no definite limitation of movement, but all movements are very slow. She puts her forefinger to the nose with exceeding slowness, and with irregular to-and-fro movements of the arm. It seems as if she were unable to sustain any muscular effort. There is no wasting of this arm, no rigidity or contracture, but there are occasional irregular muscular jerkings, chiefly in the biceps. There is some incoördination of both arms and legs.

*Sensory Functions.*—The anæsthesia has spread to the left side, so that the face, scalp, and outer side of the left leg are now the only normal parts (*vide* fig. 5). The anæsthesia and analgesia are absolute; there is complete loss of pallæsthesia over the same areas; there is complete loss of muscular sense in all its forms. She cannot feel the muscular contraction when faradism is applied so strongly as to tetanise the whole limb; she cannot tell in what position the limbs are placed, nor say when they are being moved. This loss of sense of position corresponds to the anæsthesia of the skin, it does not involve the right thumb or the left toes, in which there is no cutaneous anæsthesia. There is also complete astereognosis in both hands. A needle passed through the left supinator longus causes no pain and draws no blood.

*Reflexes.*—The tendon reflexes are brisk in the left arm, less so in the right; the others remain as before. The plantar reflexes are extensor on both sides.

The skin lesions have not been very obtrusive; occasionally new ones appear; these are invariably preceded by a small papule, which slowly increases during one or two days; it is raised above the general surface of the skin and quite painless. It then breaks open and leaves a little sore. The position of these lesions bears no relation to the bandages which are kept applied.

October 21.—For the second time since admission she has developed a functional aphonia, as a sequel to a follicular tonsilitis. Though she cannot speak, to order, above a whisper, yet she sometimes, as the sister and nurses say, forgets her aphonia and speaks quite naturally. Similar facts have been noticed about her paralysis. Thus she has once or twice either buttoned or unbuttoned her night-dress, which she says she cannot do, and then has gone and asked a neighbouring patient to do it for her.

Twice since the onset of weakness in the left arm, she has involuntarily brought the left hand to her mouth to stifle a cough with quite ordinary briskness, though the same movement, when done voluntarily, is performed, as mentioned above, with great slowness and some tremor.

Passive flexion of the right fingers still causes intense pain, in spite of the absolute anæsthesia to all other stimuli.

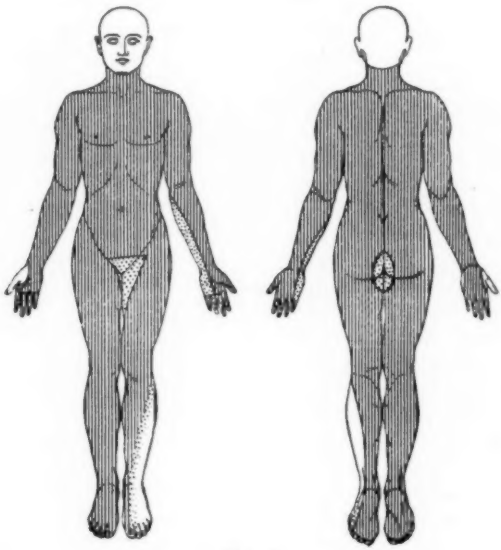

FIG. 5.

(Dr. S. A. K. Wilson.) October 15, 1905. Over dark areas, complete anæsthesia to touch, prick, temperature, vibration. Complete loss of muscular sense in same parts. Over dotted areas, a sharp pin-prick is felt as a faint touch.

November 1.—The nurse concealed a hairbrush in the patient's bed, while she was out of the ward. On coming back and getting into bed, she immediately jumped out again and said, "Who put that brush in my bed?" When nurse replied, "I thought you could not feel," the patient said she had seen it, not felt it, though it was hidden so that she could not have seen it.

November 30.—As to the sores on the skin, those on the right hip have healed, and no more have occurred during the last four

or five weeks. Those which began with a vesicle or pimple took rather a long time to heal. One of them was covered with collodion (to prevent any extraneous irritation), but it healed no quicker than the others. But on the right leg sores have reappeared. The bandage which recently has been on the left foot was taken off a few days ago, but the right lower limb has still been kept carefully swathed in lint and bandages, which have been renewed daily, and there has never been any sign that these dressings have been tampered with. In spite of this new sores have appeared within the last few days on the inner side of the right leg, and on the right instep. These are not preceded by a pimple, but are elongated lanceolate areas, about an inch long and a quarter of an inch wide. They look as if the superficial layers of the skin had been scraped off, they usually bleed a little, they heal readily; their edges are usually quite smooth, and do not seem to have been cut or hacked.

December 24, 1905.—During the last three months there has been no marked change. It has been obvious, however, that she has more or less given up hope and endeavour. Once or twice the right arm seems to have improved, but this varies. Sometimes it is possible to flex the rigid fingers with much less pain to her, but sometimes this gives her as much pain as ever. She trails the right leg uselessly behind her, but when it reaches the left foot it is neatly pulled outwards to avoid catching. She denies that she is conscious of this manœuvre. There is some improvement in the power of the left arm.

The loss of sensation remains as on the latest charts. Heat and cold are not recognised on the anæsthetic area. In all other symptoms there is very little change.

She lived about eleven months after leaving the hospital, dying eventually of intercurrent cancer, on November 19, 1906. The nervous symptoms did not alter materially, but in the course of the summer she developed an abdominal tumour. An operation was undertaken for the relief of this, but it was found to be malignant in character and insusceptible of removal.

### Autopsy.

The *post mortem* was made by Dr. Gordon Holmes and the microscopical examination of the tissues by Dr. Grainger Stewart; and I must express my thanks to them for the trouble taken by them, and for their report, which I append.

The examination had necessarily to be limited to the nervous centres.

In the *brain*, the meninges and superficial vessels were normal, nor was any evidence of disease seen externally on the cerebellum, pons, or bulb. Sections through the brain failed to reveal any definite patches of sclerosis, though there were some doubtful portions which were taken for microscopical examination.

Microscopical examination gave the following results :—

In the cortex cerebri, with Weigert-Pal stain, no changes were noted; with Nissl stain, changes were noted in the Betz cells of the motor cortex. These cells showed some chromatolysis, some displacement of the nuclei and pigmentary change. The pigment was arranged usually at one end of the cell, and was of a yellow colour. As a rule, the cell outlines were distinct. There was no evidence of a diminution of the number of Betz cells. Slight pigment changes were also seen in some of the large pyramidal cells, but these were not at all constant. With van Giesen's stain, no changes were noted.

The remaining parts of the brain were examined with Weigert-Pal stain; and (1) in the internal capsule no patch of sclerosis nor pallor was observed; (2) in the mid-brain a small patch was found lying between the pulvinar and the corpus quadrigeminum anterius of the left side; (3) in the pons and in the medulla no patches were seen; nor (4) were there any in the cerebellum. Lastly, the optic tracts showed no pallor, but the peripheral end of one optic nerve which had been removed for examination was unfortunately lost.

The *spinal cord* showed no thickening or other disease of the meninges. The cord was small; and when a finger was drawn lightly along its surface, portions of it felt harder and firmer than the rest. On section through these firm areas, the whole cross section of the cord appeared discoloured. There was no naked-eye evidence of ascending or descending degeneration.

Microscopically, sections stained according to the Weigert-Pal method showed patches of sclerosis, in which the myelinated fibres failed to take the stain scattered throughout the cord. In the cervical, dorsal, lumbar, and sacral regions, these patches involved both the grey and the white matter. In addition to these definite patches of sclerosis, pallor of the ascending and descending tracts could be seen at various places, but of very variable degree. The margins of the sclerosed patches were, as a rule, sharp and distinct, although in some places they tended to shade off into the surrounding tissue.

In detail, the position of these appearances was as follows :—

1 C. Small patch opposite either posterior nerve-root. Pallor

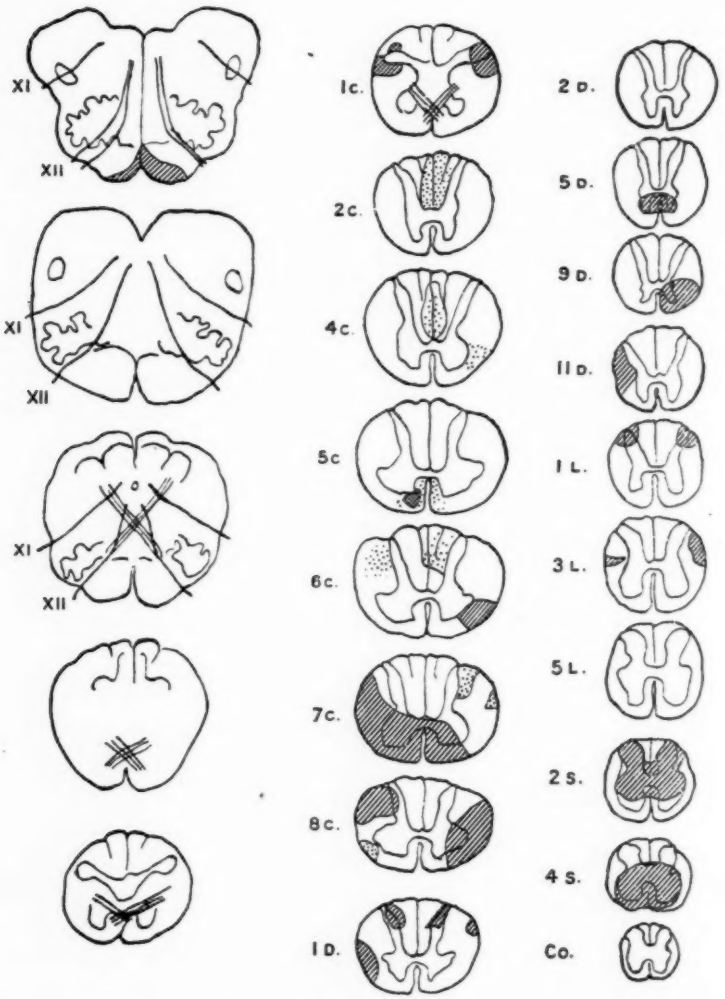


FIG. 6.

To show the distribution of the sclerosed patches in Case 2.

of postero-internal column both sides, and posterior column left side.

2 C. Similar to 1.

3 C. Pallor of posterior medium columns, slight pallor of lateral and of anterior pyramidal tracts.

4 C. Pallor of posterior median columns, of left pyramidal and anterior pyramidal tracts.

5 C. Patch in right anterior pyramidal tract; pallor of lateral and anterior pyramidal tracts.

6 C. Patch extending from external corner of left anterior horn to periphery; slight pallor of both lateral columns and of left postero-external column.

7 C. Large patch involving anterior columns and inner half of left anterior horn, right anterior horn, and anterior two-thirds of right lateral column.

8 C. Large patch involving anterior two-thirds of left lateral column and outer part of left anterior horn; patch at right posterior root; pallor of right lateral column.

There was pallor of the lateral columns throughout the dorsal regions, and, in addition, at the following levels:—

1 D. Patch extending from right anterior horn to periphery; small patch on periphery of left side of cord external to left posterior root.

5 D. Patch lying between anterior horns.

9 D. Patch in left anterior column and left anterior horn.

11 D. Patch involving right lateral column.

1 and 5 L. Pallor of anterior and lateral pyramidal tracts, and of posterior columns on right side.

2 S. Large patch involving posterior parts of anterior horns and the grey matter behind.

4 S. Large patch in same position but involving all the anterior horns.

The condition of the anterior horn cells was examined with Nissl stain, and gave the following results: in the *lumbar* region there were very few normal cells. From many cells the granules had practically disappeared, so that the cell had a homogeneous appearance. In others the granules were collected together at the edges of the cell, while in some they had disappeared, and instead of them there could be seen clumps of yellow pigment occupying part or whole of the cell.

The nuclei were placed as a rule in the centre of the cells, but sometimes eccentrically, and rarely they were hard to distinguish. The nucleoli were as a rule well stained. In the *cervical* and

*dorsal* regions similar changes were seen, though of a less marked character.

Stained with van Giesen's stain, definite changes were made out only in the sclerosed patches. Here the sections were stained darker, and there was a dense network of neuroglial tissue with very few nuclei. The margins of the patches did not appear so distinct as with Weigert-Pal staining.

The *posterior root ganglia* were examined with Nissl and with van Giesen stains. In certain of the cells some pigmentary change was noted, but this was not general and otherwise nothing abnormal was made out.

Some of the white scars from the *skin* were examined with van Giesen's stain. There appeared to be an increase in the fibrous tissue, but otherwise nothing abnormal was noted.

PLATE I.

To show the position and form assumed by the rash in Case 2 (*cf* p. 349).

PLATE I.

# THE PATHOLOGICAL CHANGES IN THE CENTRAL NERVOUS SYSTEM IN EXPERIMENTAL DIPHTHERIA.[1]

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THIS research was commenced in 1904, but circumstances have interfered with the completion of the work and its publication till the present date. A large amount of literature exists concerning the pathological changes occurring in the central nervous system in diphtheria. It is not proposed in the present communication to give a detailed account of this literature, because a complete historical survey of the subject will be found in a paper by Rainy [17], published in 1900. The net result obtained from a perusal of this literature was that some observers maintained that important pathological alterations *did* occur, while other equally competent observers were unable to find such changes. In 1902 one of us (C. B.) [6] examined the central nervous system in eleven cases of acute diphtheria. In all eleven cases acute degenerative changes were found in certain nerve-cells of the medulla oblongata, the nucleus of the vagus being especially affected. The vagus nerve itself was normal in every case. Other cases have likewise been described, and are referred to in the Bibliography.

[1] The expenses of this investigation have been in part defrayed by a grant from the British Medical Association.

From the experimental point of view the most important work has been done by Crocq [7] (1895), Mouravieff [14] (1897), and Rainy [17] (1900). These experimenters produced diphtheria in rodents, and described acute degenerative changes in the nerve-cells of the central nervous system. Martin [12] was unable to find changes in the central nervous system, but his work was done before the present methods of staining became known. He obtained paralysis of the limbs in the monkey by injection of diphtheria toxin. It may be here mentioned that it is in the *acute stage* of diphtheria that the degenerative changes in the central nervous system are likely to be found, and not in the *paralytic stage*, or at all events not to the same extent. This is exactly analogous to what obtains in the case of fatty degeneration of the heart, which is most extensive in fatal cases in the acute stage, but is only relatively slight or absent in cases surviving this stage. As Ehrlich [10] maintains, the symptoms in the acute stage are probably due to the action of *toxin*, whilst those in the paralytic stage, depending on peripheral nerve degeneration, are due to the action of *toxone*. This subject, however, cannot be regarded as settled, because Arrhenius and Madsen [1] have brought forward evidence to show that the diphtheria toxin is a homogeneous poison.

It seemed to us that it would be of interest to experimentally investigate the changes in the central nervous system of the monkey, on account of its close resemblance to that of man. At the same time we have repeated Rainy's experiments.

Briefly stated, our results confirm those of Crocq, Mouravieff, and Rainy, and in addition we have found that the nerve-cells of the monkey are likewise susceptible to the action of diphtheria toxin, and that the medullary centres of this animal are more definitely affected than are those of the rabbit.

## Method.

In all, four rabbits and three monkeys were inoculated with diphtheria toxin. This was prepared by growing

virulent diphtheria bacilli, obtained from a well-marked case of diphtheria, in beef broth, to which 2 per cent. of peptone was added, and which was rendered slightly alkaline by the addition of normal soda solution. After allowing the bacilli to grow for fourteen days, the broth was filtered through a Berkefeld bottle and bottled.

The brain, spinal cord, and heart were removed from the animals as soon after death as possible. It was not always possible to remove the organs immediately after death because some of the animals died during the night. Rainy [17] showed, by leaving the dead bodies of rabbits in cages at the room temperature for various lengths of time, that up to twenty hours no perceptible change was to be found in the nerve cells of the spinal cord, but that after this time *post-mortem* changes began to appear. We have accepted this statement, and therefore, since we could be quite certain that in no case more than thirteen hours had elapsed between the time of seeing the animal last alive and finding it dead, and probably most of the animals died in the early morning, we have not scrupled to include all the animals in the description of our experiments.

The central nervous system was hardened in 5 per cent. formalin, embedded in paraffin, cut, and stained by Nissl's method. The parts so treated were the cervical and lumbar enlargements, the whole of the medulla, pons, and mid-brain, a portion of the mid-dorsal region of the cord, pieces of the cerebral and cerebellar cortex, and certain spinal ganglia. The portions of the cord and the medulla were cut into blocks about a quarter of an inch long before embedding; the blocks were numbered and cut into sections (5-7$\mu$ thick) from below upwards. Six or more adjacent sections were mounted on a slide; about thirty sections were then discarded, and another six mounted. In this way an ascending series of sections was obtained. The sections were fixed on the slides in the usual way before staining. The heart was hardened in Müller's fluid and stained with osmic acid.

Each section was carefully examined and a description written. It seems unnecessary, however, to describe every

section in this paper, because the space occupied by such a procedure would be too large, and it would involve a considerable amount of repetition.

The central nervous system of a normal rabbit was examined as a control in the same manner.

## Results of Experiments.

### *Summary of Pathological Changes.*

The changes in the nerve-cells are as a whole slight, but here and there are cells showing gross changes.

*Slight changes.*—These consist of chromatolysis and slight swelling of the nucleus, which loses its well-defined outline and may become a trifle eccentric. The chromatolysis is central: that is, the process of breaking up of the granules begins round the nucleus and spreads to the periphery, so that the centre of the cell is occupied by a very fine granular *débris*, in which floats a more or less cloudy nucleus, and the periphery by more or less normal granules. Eventually the peripheral granules become powdery, and the process spreads into the dendrites.

*Gross changes.*—These consist of a swelling of the whole cell in various degrees, and a further change in the position of the nucleus. The swelling as a rule is not sufficient to make the cell globular, but enough to cause deformity. In the less marked cases the usual concavities between the processes of the multipolar cells are not so well marked as they should be. In more marked cases these concavities may become convexities, and the whole cell is more or less globular. The cell processes in these cases appear to have disappeared. The nucleus may be so eccentric as to lie against the side of the cell, and even to form a local bulging at that spot.

As stated above, the degenerative changes are as a whole slight, but occasionally cells showing gross changes are seen. These changes are found in the spinal cord, medulla, pons and mid-brain. The cerebrum, cerebellum and spinal ganglia were in all the cases normal. In the rabbits the changes appeared to be most marked in the lower regions of the

cord, and to diminish in extent upwards. In the monkeys they were more equally distributed, the cells of the medulla being relatively more affected than those of the rabbits.

The degenerate cells are scattered, and no definite focus of degeneration is to be found. In some sections all the cells appear to be perfectly normal, in others a few degenerate cells are lying alongside perfectly normal ones, and in others a large proportion of the cells appear to be changed.

Especial attention was paid to the large multipolar motor-cells. In the monkey the arrangement of the nuclei appears to be practically the same as in man, but it is different in the rabbit. In the spinal cord the changes seem to be most marked in the outer group of cells in the anterior horn. In the medulla and pons changes are found scattered about in the seventh and tenth nuclei, and to a less extent in the twelfth; the grey matter of the pons is also to some extent affected. Scattered degeneration is also found in the smaller cells.

Occasionally a marked difference between the two sides may be seen, as in Rabbit I., in which the lumbar enlargement showed marked changes on one side, while the other side was almost normal.

The epithelium of the central canal is normal in all the sections, and no vascular changes can be found anywhere in the central nervous system.

The heart in every case is markedly fatty, especially in the monkeys. The heart was examined because it is possible to form an idea of the degree of toxæmia from the amount of fatty degeneration of the heart which is present.

### Protocols of Experiments.

*Rabbit No. I.* Weight, 1,500 grammes.

| | | *Temperature.* | | |
|---|---|---|---|---|
| March 15, 1904 | ... | 10 a.m. | 103° F. | ... Intravenous injection of ½ cc. toxin. |
| ,, 16, ,, | ... | ,, | 102·8° F. | |
| ,, 17, ,, | ... | ,, | 103° F. | |
| ,, 18, ,, | | ,, | 102·6° F. | ... ,, ,, 1 cc. toxin. |
| ,, 19, ,, | ... | ,, | 101·6° F. | |
| ,, 21, ,, | ... | ,, | 97° F. | |
| ,, 21, ,, | .. | 7.30 p.m. | | ... Animal cold. |

Died during the night.

*Post mortem.*—9 a.m., March 22. (Probably less than thirteen hours after death).

*Lumbar region.*—In most of the sections are scattered cells showing chromatolysis with swelling of the nuclei; occasionally slight swelling of the cell is seen, but gross changes are mostly absent. Many sections show all the cells to be normal. The external group of cells in the anterior horn is especially affected, and in several sections a marked difference is observed on the two sides. In some sections as many as three or four cells in each anterior horn are affected, in others only one or two.

*Dorsal region.*—In most of the sections the cells are normal; here and there are scattered cells showing chromatolysis.

*Cervical region.*—This appears to be affected to about the same extent as the lumbar region. One cell is vacuolated, and several cells show distinct swelling, one or two being globular.

*Medulla, pons, and mid-brain.*—A considerable number of scattered cells show degeneration, but not to such an extent as in the spinal cord.

No change in the vessels, membranes, or central epithelium of any part of the central nervous system.

*Cerebrum and cerebellum.*—Normal.

*Spinal ganglia.*—(4th and 5th cervical), normal.

*Heart.*—Fatty degeneration: fatty granules much smaller than seen in human diphtheria.

*Rabbit No. II.* Weight, 2,130 grammes.

| | *Temperature.* | |
|---|---|---|
| March 15, 1904 ... | 103° F. ... | Intravenous injection of 1 cc. toxin. |
| ,, 16, ,, ... | 103° F. ... | Late in the afternoon the animal was paralysed in the hind legs. |
| ,, 17, ,, ... | ... ... | Animal cold. Died during the morning. |

*Post mortem.*—Immediately after death.

*Central nervous system.*—The changes here are scattered, and consist of chromatolysis. They are less marked than in Rabbit I., but have the same distribution.

*Rabbit No. III.* Weight, 1,530 grammes.

| | Temperature. | |
|---|---|---|
| March 23, 1904 ... | 103° F. ... | Intravenous injection of $\frac{3}{4}$ cc. toxin. |
| ,, 24, ,, ... | 102° F. | |
| ,, 25, ,, ... | 101° F. ... | Ears droop, looks ill. |
| ,, 28, ,, ... | 100° F. | |
| ,, 29, ,, ... | 99·8° F. ... | Tottery and wasted. |
| ,, 30, ,, ... | ... ... | Killed. |

*Post mortem.*—Immediately after death.

*Central nervous system.*—The changes here are of the slighter type as described above. They have the same distribution, and are very scattered, very many slides showing no abnormality whatever.

*Rabbit No. IV.* Weight, 1,420 grammes.

| | Temperature. | |
|---|---|---|
| March 23, 1904 ... | 103·2° F. ... | Intravenous injection of $\frac{1}{4}$ cc. toxin. |
| ,, 24, ,, ... | 102·4° F. | |
| ,, 25, ,, ... | 103·2° F. | |
| ,, 28, ,, ... | 102·4° F. ... | Intravenous injection of $\frac{3}{4}$ cc. toxin. Looks ill, ears droop. |
| ,, 29, ,, ... | 103° F. ... | Intravenous injection of 1 cc. toxin. |
| ,, 30, ,, ... | 102·8° F. ... | Intravenous injection of 2 cc. toxin. |
| ,, 31, ,, ... | ... ... | Paralysed in fore limbs more than hind limbs. Very breathless. Lifted up by the ears, and at once dropped dead from syncope. |

*Post mortem.*—Immediately after death.

*Central nervous system.*—On the whole the changes are more marked than those of Rabbit I. They are more numerous, every slide showing some degenerated cells, and they appear to be more advanced. There are many swollen cells, and a few are globular and appear to have lost their processes. Eccentricity of the nucleus is occasionally seen, but is not a marked feature.

*Heart.*—Fatty degeneration is extreme, and more marked than in any of the other rabbits.

*Monkey I.* (*Macacus rhesus*).—Weight, 2,875 grammes.

| | |
|---|---|
| October 19, 1905 ... | Intravenous injection of 1 cc. toxin. |
| ,, 23, ,, ... | ,, ,, 5 cc. toxin. |
| | (After this the animal refused food and only ate very little.) |
| October 27, 1905 ... | Can sit up, but lies huddled up in a corner; no paralysis. Last seen alive 7.30 p.m. |
| ,, 28, ,, ... | Found dead at 9 a.m. |

*Post mortem.*—9 a.m., October 28. *Rigor mortis* present. Cord and brain removed. There were hæmorrhages into the omentum, subcutaneous tissue, pericardium, lung and pleura. Some œdema of the lung and mediastinum was present. Heart very pale and fatty; kidneys very pale; liver exceedingly fatty.

*Medulla.*—There are acute degenerative changes in the medulla, especially at the upper part, in the region where such changes are most pronounced in the human subject. The degeneration is scattered, and normal and abnormal cells lie side by side. The changes consist of chromatolysis, with swelling of the nucleus and in a considerable number gross changes are seen (*e.g.*, swelling of the whole cell and eccentricity of the nucleus). The twelfth nucleus is, as a whole, normal. There is considerable degeneration in the nucleus ambiguus, especially in the inner group of cells belonging to the nucleus. Scattered degeneration occurs in the nuclei containing the smaller variety of cell.

*Pons.*—The seventh nucleus shows one or two cells in a condition of chromatolysis. Many large pontine cells show swelling and chromatolysis.

*Mid-brain.*—The oculo-motor nucleus is normal. There are a few degenerate cells at the lower part of and anterior to the iter.

*Cerebrum and Cerebellum.*—Normal. There are no vascular lesions. The epithelium of the central canal and the meninges are normal.

*Monkey II.* (*Macacus rhesus*). Weight, 2,500 grammes.

November 4, 1905 ... Intravenous injection of 3¼ cc. toxin.
,, 6, ,, ... Looks ill; refuses food. Last seen 8 p.m.
,, 7, ,, ... Found dead 9 a.m.

*Post mortem.*—Immediately. There had been bleeding from the mouth and rectum. All the organs were in a condition similar to those of Monkey I., but the changes were less marked.

*Central nervous system.*—The pathological changes are slighter and much less marked and extensive than those found in Monkey I.

There is scattered degeneration in the mid-brain, pons and medulla. The cord shows hardly any changes at all.

*Monkey III.* (*Macacus rhesus*). Weight, 2,950 grammes.

November 16, 1905 ... Intravenous injection of 2 cc. toxin.
,, 20, ,, ... Looks ill.
,, 23, ,, ... Much better; almost recovered. Intravenous injec- 5 cc. toxin.
,, 25, ,, ... Found dead, 9 a.m. Last seen alive November 24, 7.30 p.m.

*Post mortem.*—Immediately. No external bleeding. A few scattered hæmorrhages, as in Monkey I. Heart very pale and fatty. Liver very fatty.

*Central nervous system.*—The changes are practically the same as in Monkey I. The chief change is chromatolysis, but many cells show gross changes. The lumbar region of the cord appears to be more affected than the cervical, but neither shows such marked changes as are seen in the medulla.

## Conclusions.

The results of our experiments confirm those of the observers before mentioned, and also show that the monkey is very susceptible to the action of diphtheria toxin.

The medullary centres of the monkey appear to be more definitely affected than are those of the rabbit. In this respect the action of the toxin upon the monkey agrees with its action upon the human subject, in whom degeneration of the medulla is very apt to occur. We do not wish to maintain that these changes directly bring about death, nor do we go so far as some observers, who assert that they give rise to a descending degeneration in the peripheral nerves. We believe that the diphtheritic poison *directly* causes acute degeneration of certain cells of the central nervous system, and that these changes are not secondary to any disturbance in the circulation; that the poison *directly* affects the peripheral nerves, which do not degenerate simply because their central cells are temporarily affected; that the poison *directly* causes fatty degeneration of the heart, and that this does not occur merely because the medullary centres are affected.

It seems to us that these cell changes will account for the marked irregularity in the action of the heart which occurs in all cases of severe diphtheritic poisoning, and probably also for the vomiting which occurs in the early stage of diphtheria, and which closely simulates cerebral vomiting. The changes may also account for those cases of *early* muscular weakness, which come on as early as the seventh day, before nerve degeneration occurs; and on the second day in Rabbit II. of this series. It is to be noticed that in man muscular paresis in diphtheria most commonly affects the muscles supplied by the cranial nerves, whereas in the rabbit it chiefly affects those supplied by the spinal nerves. It seems, therefore, that in the higher mammals the poison especially attacks the medullary centres and the cranial nerves, and in the lower mammals the spinal centres and the spinal nerves. In all animals alike the heart muscle is directly affected by the poison, and it is this acute fatty degeneration of the muscular fibres of the heart which is the immediate cause of death in acute diphtheritic toxæmia.

Finally we beg to thank Professor Sidney Martin for permission to work in the laboratory.

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# Reviews.

*The Integrative Action of the Nervous System.* By C. S. Sherrington, D.Sc., M.D., Hon. LL.D.Tor., F.R.S. Constable & Co., London, 1906.

The ten lectures reproduced in this volume were delivered before Yale University as the second series on the Silliman Foundation. The first lecturer on this new foundation was Professor J. J. Thompson, whose lectures were published in the volume entitled "Electricity and Matter." Yale University is to be congratulated upon its choice, which has resulted in the production of two books of the greatest value and distinction.

This series of lectures embodies much of Professor Sherrington's own work, bringing together in one connected system the principal results of the brilliant researches reported in the many papers that have appeared in rapid succession in the Proceedings of the Royal Society and other journals. These papers have fully maintained the great prestige of the line of British neurologists, created by Sir Charles Bell and Marshall Hall, and have already secured for their author an indisputable place in the very first rank of physiologists. This book, which reveals the unity of plan underlying almost all Professor Sherrington's work, cannot fail to enhance his great reputation, and to make his work known to a wider circle of readers. The general conception and aim of the book render it unique, and mark a distinct step forward in the long siege of that crowning problem of the biological sciences, the functions of the central nervous system of man. During the last fifty years the stream of publications on this subject has swelled at a constantly increasing rate, and there has been no lack of well-written books reviewing the work done and bringing together its principal results. But no one of these has been centred upon the essential function of the nervous system, the integration of the organism. They have discussed the intimate nature of nervous processes in the structural elements; they have exhibited the anatomical relations of parts, both large and small; they have shown that

certain parts are essential to certain forms of bodily behaviour or to certain modes of consciousness; but they have left in almost unrelieved darkness the problem—how, given the primary capacities of nervous tissue and the anatomical relations of its parts, do the tissues, so endowed and so arranged, effect the integration, the functional unification or harmony, of the parts of the organism, making an individual of what, apart from them, would be a mere aggregation of organs? Sherrington has clearly seen that this is the central problem to which all the others are but subsidiary. And he has seen that a way to the understanding of this supreme function of the nervous system can be opened only by a thorough study of the co-ordinations of movement effected by the functional dispositions of the spinal cord. For it has become constantly more clear that, just as, phylogenetically, the brain is a late outgrowth from the spinal axis, so its functions are those of the spinal axis carried to an indefinitely higher degree of complexity and nicety; and it seems clear that this will remain true, even though the processes of the cord should prove to be capable of being fully described in chemical and physical terms, while those of the brain are found to involve activities of a higher category. All study of the brain then must be, in a sense, premature, a mere irregular skirmishing, the results of which must remain as accumulated data that cannot be fully applied and harmonised until the victorious army of science, pressing on along the line marked out in this volume and having completed the conquest of the cord, shall enter the citadel by its main gate, prepared to penetrate its mysterious ways in the light of the knowledge gained in the assault upon the outworks.

The larger part of the book is, then, devoted to the study of the reflex actions manifested by the "spinal animal." Following on these purely spinal lectures are two which discuss the modifications of spinal functions determined by the presence of the brain; and the final lecture shows how the study of sensation and perception must be brought to our aid when we attempt to push up from the spinal cord to the brain, and indicates the way in which neurology and psychology may be brought into effective co-operation in their march towards their common goal, the understanding of the behaviour of living organisms.

The integrative action of the spinal cord is regarded as consisting in the co-ordination of movements in response to sensory stimuli, and this co-ordination is regarded as the simultaneous and successive compounding of simple reflexes. The simple

reflex, we are warned, is, like the simple sensation, a fiction; for in the intact animal there is no such independence of parts as is implied by the conception of a simple reflex. Nevertheless it is a useful and, therefore, legitimate fiction, and the book opens with a discussion of the characters by which a simple reflex is distinguished from simple conduction through a motor nerve; these are enumerated in the following list: (1) longer latency or lost time; (2) after-discharge; (3) rhythmic discharge; (4) greater independence of the stimulus in regard to intensity of motor effect; (5) resistance to the passage of the impulse and summation of stimuli; (6) irreversibility of conduction; (7) fatigability; (8) greater variability of response; (9) facilitation, inhibition and shock; (10) greater sensitivity to chemical agents, *e.g.*, oxygen and various drugs.

Sherrington holds that the *synapse* plays an important part in the determination of all the peculiarities of the simple reflex enumerated above. It is probable that the conception of the synapse will be found to be a great landmark in the history of the physiology of the nervous system. The term "synapse" was introduced by Foster and Sherrington in 1897 to fix the functional conception of the structure which forms the junction of two or more neurones. The name and the conception have rapidly gained favour in this country, although up to the present time they are, apparently, little known beyond its borders.

From the time of the discovery of the nucleated cell-bodies of the nerve-cells (the so-called ganglion-cells) until the middle of the nineties, almost all writers attributed to them all the features in which conduction through grey matter was found to differ from the conduction of the impulse in peripheral nerve-fibres. The implicit argument ran—the nervous system consists of fibres and ganglion-cells, therefore all that is not due to fibres must be attributed to the cells. Then came the histological discovery that cell-body and fibre together make up the cell or neurone, and that each cell has apparently no substantial continuity with others. Unfortunately the neurone controversy has raged, and still rages, round the anatomical question of continuity or discontinuity of substance. Thus Pflüger in a recent paper (*Arch. f. ges. Phys.*, Bd. 112, 1906): "Unsere Aufgabe ist also zu entscheiden ob die Leitung von einer Nervenfaser auf eine Zelle oder von der Zelle auf die Nervenfaser durch Contakt oder Contiguität sich vollzieht." And Pflüger, like Bethe, Lewandowski, and many others, finding good grounds for believing that continuity is the rule, regards the neurone-theory as disproved, and

treats it scornfully as an exploded and pernicious fallacy. But it is only a very narrow view of the neurone-theory that regards its validity as turning upon the question of *Contiguität oder Continuität*, if indeed this question is not a mere mis-statement of the essential problem; for no one has seriously maintained that a vacuum intervenes between functionally related cells. Even though, as Bethe, Held, and others claim to have proved, continuity of substance between neurones is histologically demonstrable, the neurone-theory, not as a merely anatomical but as a physiological conception, remains unaffected, for the essential problem is—Is the process of transmission from neurone to neurone similar to simple conduction along a nerve-fibre, or is it a very special process conditioned by peculiarities of structure or substance at the junction?

Sherrington's doctrine of the synapse replies affirmatively, and, as we have seen, attributes very important functions to the synapse. The grounds which justify this attribution are many and various, and of constantly increasing weight, and, though they cannot be said to be absolutely conclusive, they would justify the synaptic-neurone as a working hypothesis, even though a histological continuity of substance of nerve-cells as complete as that demonstrated by Apáthy, Held, and others in certain instances, could be shown to be universal. But recent histological research has yielded results which, far from being adverse to the synaptic-neurone conception, strongly favour it, although Sherrington does not call them up in evidence; he is content to take his stand upon the purely physiological evidence, and makes, from that standpoint, some interesting speculations as to the constitution of the synapse. He suggests that it may be essentially a surface of separation, constituting a physical membrane between the protoplasms of the functionally connected cells. He writes: "If it (conduction) is a physical process, the intercalation of a transverse surface of separation or membrane into the conductor must modify the conduction, and it would do so with results just such as we find differentiating reflex-arc conduction from nerve-trunk conduction" (p. 17). Here, no doubt, Sherrington follows sound method in assigning to the synapse the simplest possible constitution that will account for the peculiarities of central conduction as he has studied them in the cord; but many considerations seem to indicate that the synaptic processes are more complex, and involve special chemical as well as physical, changes. The present writer, approaching the subject from the psychological side, argued,

as long ago as 1897, that not only the peculiarities of conduction of the spinal cord, but also those of the cortical grey matter, especially the great susceptibility to derangement by anæsthetic drugs and the determination of all the elementary affections of consciousness, must be attributed to peculiarities of constitution of the neurone junctions, and inferred that in that case we are bound to regard these junctions as the seats of chemical processes taking place in intercellular substances of very highly specialised constitution (*Mind*, N. S., vol. vii.). And, in the following year, Nissl and Bethe produced independently histological evidence of the presence of intercellular substances (the *Fibrillar-gitter* on the *Golgi-netz* of Bethe, the *Neuropil* or *zentraler Grau* of Nissl), and they both inclined to attribute to them very special functions. Some of the peculiarities which Sherrington himself attributes to the synapse of the simple reflex-arc can with difficulty be accounted for by the assumption of a mere surface of physical separation, more especially the profound modification of its functioning by fatigue, oxygen, strychnine and tetanus toxin.

However this may be, the doctrine of the synapse becomes in Sherrington's hands the key to the understanding of the integrative functions of the nervous system, and the success with which it is thus applied throughout these lectures is its best justification. If we do not accept it, we must either give up the problem, or be content to fall back on the old-fashioned way of "explaining" the functions of the central nervous system by invoking mysterious automatic powers of "centres" and "ganglion-cells," a way which consists in postulating in each centre or cell the power of producing whatever effect is observed, and which almost amounts to making each of them an independent centre of "free will" or spontaneous new beginnings.

After treating of "the simple reflex," Sherrington goes on to consider the coördination of reflexes, which consists in the coöperation of reflexes of allied or harmonious tendencies, and the prevention of simultaneous activity of reflex-arcs of antagonistic tendencies. Here two great principles come into view, that of the common path and that of reciprocal inhibition.

"While the receptive neurone forms a private path exclusively serving impulses of one source only, the final or efferent neurone is, so to say, a public path, *common* to impulses arising at any of many sources of reception. A receptive field, *e. g.*, an area of skin, is analysable into receptive points. One and the same effector organ stands in reflex connection not only with many individual receptive points, but even with many various receptive

fields. Reflexes generated in manifold sense-organs can pour their influence into one and the same muscle. Thus a limb-muscle is the *terminus ad quem* of many reflex-arcs arising in many various parts of the body. The motor nerve is a path common to all the reflex arcs which reach that muscle;" and again, "Before finally converging upon the motor neurone the arcs converge to some degree. Their private paths embouch upon *internuncial* paths common in various degree to groups of private paths. The terminal path may, to distinguish it from internuncial common paths, be called *the final common path*. The motor nerve to a muscle is a collection of final common paths" (p. 116). This is the principle through which the simultaneous coöperation of reflex tendencies is effected; in the light of it the phenomena of *Bahnung* or facilitation, and of simultaneous spinal induction and re-enforcement, are seen to be of the same nature as the summation of effects of sensory stimuli applied in succession to the same receptive point, which in turn is rendered intelligible by the doctrine of the synapse with its variable threshold of resistance.

Sherrington points out that the wide prevalence of the arrangement known as the common path seems to justify us in believing that in all sensory and motor nerve-fibres the conduction-process is of the same nature; for, since stimulation of sense-organs of widely different functions may give rise to excitations which issue conjointly along the same final common path, it is difficult to suppose that the conduction-process initiated in each sense-organ has its own specific nature, as Hering, Head, and others have maintained. The sorting or re-grouping of afferent impulses in the spinal cord, which Head has demonstrated, is certainly well explained by his theory of intramedullary specific receptors (*Brain*, 1906), but it is necessary to remember that it may be the afferent nerve-fibres, rather than the impulses alone, that are re-grouped within the cord.

The other great integrative principle, that of reciprocal inhibition, is more obscure. It seems to underlie not only the harmony of action of spinal reflex tendencies, but also that concentration of the whole organism upon one action at any one moment which characterises all the higher organisms, and is the essential mark of individuality or complete integration of the vital unit. But as to the mode of effectuation of such integrative inhibition no agreement has yet been reached. Sherrington looks with favour on the hypothesis of inhibition by drainage, put forward by the present writer (*Brain*, 1903), but raises two objections which are, perhaps, not insuperable. Of these two objections, the more

serious, in his opinion, is the fact that this hypothesis "seems to sever central nervous inhibition from other forms of inhibition met peripherally in the heart, blood-vessels, and viscera, rather than to connect it with-them, and it appears to me unlikely that in their essential nature all forms of inhibition can be anything but one and the same process" (p. 203). In answer to this objection, it might be pointed out that the organs which are inhibited by impulses passing down efferent nerves are those which, like the heart and the bowel, seem to be capable of automatic or locally originating movement, and that, therefore, it is impossible they should be inhibited and controlled, like skeletal muscles, by the mere cutting off of impulses at the spinal centres; and further, if this automatic movement is due to local nervous mechanisms, as some authorities still believe, then inhibition of it may ultimately be shown to conform to the drainage type; but if, on the other hand, it is myogenic, then these tissues are, *ipso facto*, fundamentally different from the skeletal muscles, and, since inhibition of them is then an intramuscular process, whereas inhibition of skeletal muscles is an intranervous process, the two kinds of inhibition can hardly be "one and the same process." Again, all these peripherally inhibited organs belong to the visceral sympathetic system, which bears many signs of being a phylogenetically older system than the skeletal system, and such a primitive system may well continue to conform to principles of action which would have proved cumbrous and inadequate if adopted for the control of the skeletal system.

The second objection may be stated as follows: Inhibition of a reflex contraction differs from the mere cessation of stimulation of the arc in two ways, (1) cessation of stimulation is followed by an after-discharge, *i.e.*, the reflex contraction only gradually remits, but on inhibition the contraction is suddenly cut short; (2) there occurs *successive spinal induction*. This latter phenomenon Sherrington has described and carefully studied, and he suggests that it may be a process similar to successive visual induction. When, of two arcs in relation of reciprocal innervation, both are stimulated, but one, B, is more strongly stimulated than the other, A, the former, B inhibits A; then, if the sensory stimulation of B ceases while that of A continues unchanged, the contraction of the muscle-group of A at once returns and becomes more intense than it was before B was stimulated; *i.e.*, the inhibition of A by B is immediately followed by augmentation of response of A to stimulus of unchanged strength.

On this Sherrington remarks that "the diversion of the influence of $a_1$ (the afferent neurone of arc A), which constitutes inhibition (according to the drainage hypothesis), does not suggest a reason for the superactivity (successive induction) in A consequent on the inhibition." But it may be suggested that the cutting short of the after-discharge of B gives us the key to the explanation of the augmented activity of A; for some such effect is demanded by the drainage hypothesis, and though the augmentation of the effect may seem too large to be accounted for in this way, we have to remember that, as Sherrington writes, "each reflex movement must of itself generate stimuli to afferent apparatus in many parts and organs—muscles, joints, tendons, &c."; and if there is any truth in the drainage hypothesis, all these excitations, secondarily resulting from the stimulation of B, must, when the excitation of A becomes dominant again, contribute to its augmentation. That is to say, successive induction is an effect that is not only not incompatible with the drainage hypothesis, but is demanded by it.

There is, further, a strong argument in favour of the drainage hypothesis suggested by Sherrington's observations, though not pointed out by him. "A single receptive point may play reflexly upon quite a number of different effector organs. It may be connected through its reflex path with many muscles and glands in many different regions. Yet all its reflex-arcs spring from the one single shank or stem, *i.e.*, from the one afferent neurone which conducts from the receptive point at the periphery into the central nervous organ." Now, on stimulation of this one receptive point with its single afferent neurone, not all these effector organs necessarily or usually come into play; the same stimulus to the same receptive point may at one time bring into play one muscle-group, at another time another and very different, sometimes even an antagonistic, muscle-group. That is to say, the "impulse" or the innervation current may, after traversing the common shank, be diverted into any one of a number of different efferent channels, according to the conditions of those channels at the moment of incidence of the stimulus. "The momentary condition of any cell-chain is in part a function of the condition at the moment of all the other cell-chains with which it is connected" (p. 271); and again, "the high variability of reflex reaction . . . . . , though often attributed to general conditions of nutrition, or to local blood-supply, &c., seems far more often due to changes produced in the central nervous organ by its own functional

conductive activity apart from fatigue. This functional activity itself causes from moment to moment the temporary opening of some connections and the closure of others" (p. 232); and again, "the direction of the stream of liberation of energy along the pattern of the nervous web varies from minute to minute" (p. 233). This variation of the path taken by an afferent impulse, according to the variations of condition of the several alternative paths at various times possible for it, is obviously a principle of no less importance than that of the common path and that of reciprocal inhibition, and, like them, it must be of increasing importance as we ascend to successively higher levels of the central nervous system. Now, if the drainage hypothesis of inhibition is rejected, this variability of path remains as a third principle independent of the others and utterly unexplained. But, if we accept that hypothesis, we see at once that this variability is to be explained by the application of the same principle which gives us the drainage hypothesis, the principle, namely, that the nervous impulse follows the path of least synaptic resistance, and that these synaptic resistances are lowered in proportion to the state of excitation of the neurones between which they constitute at once the connections and the barriers. This, which might be called the *principle of excitatory variation of synaptic resistances*, would be a higher principle from which the two subsidiary principles, that of inhibition and that of the variability of reflex response, might be deduced; and it would be one which might carry us far in the elucidation of the integrative functions of the brain itself.

After discussing, and richly illustrating from his own experience, these and many other features of the spinal reflexes, especially fatigue, shock, tonus, the influence of strychnine, and the adaptive grading or subordination of reflexes which secures precedence for those initiated by stimuli of greatest urgency, *e.g.*, hurtful stimuli, Sherrington goes on to discuss the influence of the brain, especially of the motor cortex, upon the spinal coördinations. We are led to see that, just as the simple reflex of the cord is but a fragment of a larger functional spinal system, so the larger spinal systems are but fragments of still larger coördination systems, working on the same principles, but on a larger and more complex scale, in the motor cortex.

Sherrington's studies of "decerebrate rigidity" have led him to recognise a most important distinction between two systems of skeletal musculature and their controlling spinal mechanisms—"One system exhibits those transient phases of heightened re-

action which constitute reflex movements; the other maintains that steady tonic response which supplies the muscular tension necessary to *attitude*" (p. 302); they are the phasic and the tonic reflex systems. "Decerebrate rigidity exhibits reflex excitation in these very groups of muscles which the local reflexes and the motor cortex, when stimulated, excite but little," namely, the muscles of the tonic system, which, in the main, consists of the great extensor groups that maintain posture by means of a reflex tonus, of which the stimulus is the force of gravity acting through local deep afferent neurones of the proprio-ceptive system and those of the otic labyrinth. And the contractions of this tonic system are "inhibited by the overwhelming majority of local spinal reflexes and of reactions from the motor cortex." This important distinction, according to which the skeletal musculature falls into two large systems of opposed tendency, thus reveals the principle of reciprocal innervation at work upon a larger scale and a higher plane than in the cases of simple pairs of antagonistic muscle-groups.

Sherrington has introduced the terms proprio-ceptive and extero-ceptive to denote the sense-organs whose adequate stimuli come from within and from without the body respectively; the former actuate, in the main, the tonic system, the latter the phasic system. And he distinguishes by the name intero-ceptive a third group of sense-organs, those which lie in the membrane lining the alimentary canal, and which are characterised by their intimate reflex relations with the visceral musculature.

The sense-organs of the extero-ceptive field are of two classes, broadly distinguishable as the less specialised organs normally stimulated by objects in contact with the surface of the body, and the special sense-organs of the head, whose stimuli normally come from distant objects, and to which Sherrington therefore gives the name distance-receptors. The great brain has been evolved in connection with these distance-receptors, whose functions have constantly acquired, in the phylogenetic process, an increasing predominance over those of the non-projicient receptors. (It may be called the ganglion of the distance-receptors, while the cerebellum may be called the "head-ganglion of the proprio-ceptive system.") We are then shown that, while the extero-ceptors of these two classes actuate, in the main, the same muscle-groups, they do so in two very different modes. While the stimulation of the non-projicient receptors produces local segmental movement, such as flexion of one joint or limb, "the action of these latter (the distance-receptors) is characterised

by tendency to work or control the musculature of the animal as a *whole*—as a single machine—to impel locomotion or to cut it short by the assumption of some total posture, some attitude which involves steady posture, not of one limb or one appendage alone, but of all, so as to maintain an attitude of the body as a whole." "The skeletal musculature is treated practically as a *whole*, and in a manner suitably anticipatory of a later event."

Thus the evolution of the distance-receptors and their arcs brings about a great step in the integration of the organism. Their integrative action is effected by means of extensive internuncial paths that are common to several receptors, and that issue in the final common paths of the skeletal system. Of these internuncial paths the pyramidal tract is the principal. It is through them that the distance-receptors exert their preponderant influence over the whole motor system, and become the principal initiators of movement. But "the distance receptors integrate the individual, not merely because of the wide ramification of their arcs to the effector organs through the lower centres; they integrate especially because of their great connections in the high cerebral centres" (p. 353).

The distinction between the proprio-ceptive tonic system and the extero-ceptive phasic system, and the conception of the cerebellum and the cerebrum as the ganglia of the tonic and phasic systems respectively, lead Sherrington to a new and profounder explanation of the difference between cerebellar and cerebral influx, so brilliantly discerned by Dr. Hughlings Jackson thirty-five years ago, and since abundantly confirmed by experimental and clinical observations (summarised by Sir Victor Horsley in the Hughlings Jackson lecture, *Brain*, 1906). Sherrington shows that decerebrate rigidity does not depend upon the cerebellum, and that the tonic character of the spasms of cerebellar epilepsy and the phasic character of cerebral epilepsy are due, not to any radical difference between the cerebellar and cerebral discharges, but rather to the fact that the cerebellum, the ganglion of the proprio-ceptive system, discharges itself mainly upon the tonic extensor system of spinal reflex arcs, while the cerebrum, the ganglion of the distance-receptors, discharges itself in the main upon the phasic flexor system of arcs and muscles.

It is worth noting that these illuminating distinctions suggest an explanation of the phenomena of decerebrate rigidity in terms of the drainage hypothesis, and so remove what has seemed one of the principal difficulties in its way. The sense-organs and arcs of the proprio-ceptive system, being constantly

stimulated by the bodily effects of gravity, tend to maintain a postural tonus, their influence being directed upon the tonic (in the main extensor) system of skeletal muscles. The two extero-ceptive systems, the non-projicient and the distance-receptors actuating the phasic system, are in relation of reciprocal innervation with the proprio-ceptive system; in the intact animal the distance-receptors, constantly stimulated through eye, ear and nose, constantly exert a powerful excitatory influence upon the phasic system, and, in doing so, divert more or less the influence of the proprio-ceptive stimuli from the tonic system; that is to say, they inhibit its tonus by keeping the flexor final common path in excitation, *i. e.*, more open than the extensor paths. But when the internuncial paths, by which these effects are produced, are broken across, or the cerebral hemispheres are removed, the tonic proprio-ceptive system is antagonised only by the occasional, local, and relatively weak flexor tendencies of the stimuli falling on the non-projicient receptors; and, although strong local stimulation of this system will produce, while it lasts, local remission of the extensor rigidity (p. 295), the stimuli falling on these extero-ceptors are in the main inadequate to cope with the powerful, constantly maintained, proprio-ceptive influences, which thus keep open the final common paths of the extensor groups, with the correlative effect of closing the flexor paths.

The same distinctions seem to suggest an explanation of the effects of strychnine on the spinal cord. Sherrington shows that in the spinal animal reciprocal innervation is broken down by strychnine, and that under strychnine the inhibition of a muscle-group, normally produced by stimulation of the afferent nerve that excites its antagonist, is apt to be converted into excitation, so that antagonistic muscle-groups become simultaneously excited. He adduces good grounds for rejecting the view that such an afferent nerve contains two sets of fibres, one tending to excite, the other tending more strongly to inhibit, the antagonistic muscles, and that strychnine converts inhibition to excitation by strengthening relatively the action of the former set; and he is inclined to regard the essential effect of strychnine as a breaking down of spinal resistances. But this view also he rejects on the following grounds. He takes the internal saphenous nerve, stimulation of which in the normal state of the cord always produces relaxation, *i. e.*, inhibition, of the vasto-crureus muscle, a single-joint extensor of the thigh, and he isolates the reflex by severing the nerves of adjacent parts; then "a branch of the internal saphenous nerve below the knee is also prepared for stimulation

of its central end. When this is done it is found that no change in intensity or other conditions of excitation of the afferent nerve ever provokes anything but inhibition of the extensor of the knee, but a small dose of strychnine at once transmutes the inhibitory effect into an excitation effect. Reflex contraction is obtained in place of reflex relaxation. If small doses are carefully graded, it is possible to see a state in which the reflex relaxation is diminished but not replaced by excitation. This phenomenon shows well how little competent is the view of lowered spinal resistance really to explain the action of strychnine; for at this stage the stimulated arc that normally acts on the extensor muscle by inhibition is less able to affect it than before, so that on the spinal resistance view the resistance at this stage is actually heightened" (p. 109).

This conclusion, that the spinal resistance is actually raised rather than lowered by strychnine, is only justified so long as we assume with the majority of authors (as Sherrington does here and elsewhere in these pages) that inhibition involves the transmission of some positive influence to the inhibited motor neurones; for it is this hypothetical influence which, in the experiment just described, appears to be prevented from reaching those neurones. But if we adopt the drainage hypothesis, this experiment no longer offers any difficulty to the view that strychnine produces its characteristic effects by diminishing or abolishing the synaptic resistances; and that view, when thus combined with the drainage hypothesis, seems, in fact, adequate and open to no serious objection. Thus, to take the experiment just quoted, the internal saphenous nerve must be regarded, according to the drainage hypothesis, as connected with both the flexor and the extensor motor neurones of the thigh (and the experiment shows that this is, in fact, the case),[1] but the normal resting resistance of the synapses that connect it with the flexor group is lower than that of the synapses that connect it with the extensor group. Similarly the afferent nerve, that normally produces excitation of the extensors and inhibition of the flexors, is connected with the same two groups of motor neurones, but its synaptic connection with the extensor group is normally of lower resistance than that with the flexor group. Hence stimulation of the saphenous nerve excites flexors and inhibits extensors, and stimulation of the other nerve produces the opposite effects. But if now strychnine reduces to a minimum, and so renders equal, the resistances of both sets of synapses, those on the extensor and those on the flexor neurones, stimulation of either nerve must lead to excitation

[1] See diagram, p. 201.

of both sets of muscles; and the gradual transition from the normal to the abnormal response observed in the experiment quoted above is quite in harmony with this view.

The adoption of the drainage hypothesis allows us also to apply this view of the action of strychnine to the explanation of the effects of cortical activity during strychnine poisoning. As is well known, voluntary effort at any kind of movement is apt to be unsuccessful and to result merely in an intensification of the extensor spasms; and Sherrington shows that stimulation of a spot of the motor cortex, from which inhibition of some muscle-group is normally obtained, may result in contraction of that group; *e.g.*, "when the 'face-area' of the monkey's cortex is tested by faradisation after exhibition of strychnine, the points of surface that previously yielded regularly the free opening of the jaw yield strong closure of the jaw instead. Now closure of the jaw is a movement of very limited representation in the cortex of the monkey, even of the anthropoid. On the other hand, *opening* of the jaw is always readily and regularly elicitable from a large field of the 'face-area.' And adjoining and overlapping this large area, whence steady opening of the jaw is obtained, is found an area whence, as Ferrier first pointed out, 'rhythmic, alternating opening and closing of the jaws,' as in feeding, can be evoked. Under tetanus-toxin and strychnine the whole of this combined area not only ceases to yield opening of the jaws, either maintained or rhythmic, but yields closing of them instead" (p. 295). Now the muscles and arcs that close the jaw belong to the tonic proprio-ceptive system, those which open it to the phasic extero-ceptive system (hence the larger cortical representation of the latter), and their relations to the motor cortex would seem to be typical of those of the tonic and phasic systems respectively; that is to say, in general terms, the greater number of internuncial paths descending from the cortex play principally upon the phasic system, but, as shown by the effects of stimulation under strychnine, each is connected not only with the spinal motor neurones of some one phasic muscle-group, but also, though less intimately, with the motor neurones of the tonic antagonists of that group. And if we suppose that the greater intimacy of connection with the phasic group consists in the lower normal resistance of the synapses, it follows that, when strychnine has abolished the difference of resistance between the synapses of the two groups, any voluntary effort to contract a flexor group will result in impulses which, descending in normal fashion from the cortex, will, other things being the same, pass to the tonic group as

readily as to the phasic flexor group; and, since the tonic motor path is kept in constant excitation by stimuli falling on the proprio-receptors, they will for the most part follow that path rather than the flexor path, and so will fail to produce flexion, and will but augment the intensity of the general but predominantly extensor muscular spasm.[1]

The lecture on "sensual fusion" the last of the series, opens with the profoundly interesting question, "Can we at all compare with the simultaneous coördination of the nervous factors in a motor reflex the synthesis of the nervous elements whose combination underlies a simple sense-perception?" Sherrington has sought to throw light on this question by investigating the facts as presented in one of the simplest possible instances, namely, in the case of the binocular fusion of the effects of similar stimuli applied to corresponding areas of the two retinæ. By means of a most ingeniously designed piece of apparatus, he was able to make observations of a more exact and decisive character than any previously made in this field. The principal result is the refutation of the view, so long and so widely held among physiologists and psychologists alike, namely, the view that this binocular fusion implies and depends upon the convergence of the fibres from each pair of corresponding retinal elements upon a common central path, fibre, cell, or centre, a view which the facts of semi-decussation of the optic tracts have quite illogically been held to justify. "The experimental results go to disprove the existence of any such fusion or interference between the apsychical or even the subperceptual events arising from corresponding retinal points." There was already evidence to this effect, sufficiently conclusive to those who were familiar with it, but the new evidence provided by these more exact experiments is more convincing and should serve to shake the widespread prejudice in favour of the alternative view, which is a special application of, and logically implies, the untenable doctrine of the *sensorium commune* or the "pontifical cell."

As regards only one point can exception be taken to Sherrington's formulation of his conclusions. He writes: "Our experiments show, therefore, that during binocular regard of an objective image each uniocular mechanism develops indepen-

[1] On writing this passage it seemed to me so highly probable that the action of strychnine is to diminish or break down the synaptic resistances, and that the action of chloroform (as I suggested in 1897) is to increase these resistances, that I wrote to Prof. Sherrington to enquire if he had noticed any indications of direct antagonism between the influences of the two drugs. His answer was that he had recently had occasion to observe an "exquisite antagonism" between them.—[W. McD.]

dently a sensual image of considerable completeness. The singleness of the binocular perception results from union of these elaborated uniocular sensations. The singleness is therefore the product of a synthesis that works with already elaborated sensations contemporaneously proceeding." This phraseology implies that in binocular vision two sensations come into existence independently, and that then by a further process, succeeding in time to that which produces the two sensations, they are fused together to form a single sensation or percept; and the terms, psychical fusion and synthesis, so commonly used and almost indispensable in psychology, seem to imply this kind of process. But they can only be legitimately used if it is made clear that they are not to be taken in this sense. We have no evidence that the sensations are first separate and then fuse together. What the evidence shows is that the two brain-processes (including those parts which are the immediate excitants of sensation), initiated by simultaneous stimulation of corresponding points of the two retinæ, run their course in separate anatomical elements; and we ought rather to say that these two separate processes coöperate in determining the one psychical resultant, the affection of consciousness that we call the binocular sensation. If we state the facts in the other way, we imply that the uniocular sensations, which we describe as fusing together, exist before this fusion as unconscious sensations, and we then find ourselves groping in the obscure region of the unconscious psychical, committed to the doctrine of unconscious modifications of consciousness, "the sovereign recipe for turning what might become a science into a tumbling-ground for whimsies" (James).

The distinction here insisted on is no doubt a subtle one, but it is nevertheless important; for this case of binocular fusion of effects of stimuli is, as Sherrington rightly sees, but a specially clear and simple case of that composition of the psychical effects of many simultaneous brain-processes which is an ever-present condition of our consciousness; and our attitude here must determine our treatment of the psycho-physical problem in all its aspects. Sherrington sees positive justification of his mode of statement in the phenomena of Fechner's paradoxical experiment; for the explanation of these that he offers is based upon the assumption of the separate existence of the uniocular sensations, followed by their fusion; but the present writer has shown (*British Journal of Psychology*, vol. i.) how these curious effects may be explicable by reciprocal inhibition between the afferent neurones of the two retinæ in a manner that does not commit us to the postulation of unconscious uniocular sensations.

We seem, in fact, justified in adopting as our fundamental psycho-physical postulate the unity of the psychical effects of all sensory stimuli affecting the one organism, and in seeing, as the problem requiring a psychological solution, not the fusion of these effects, but the possibility of introspective analysis of the combined psychical resultant. And the important lesson that Sherrington bases on the results of these experiments on sensual fusion is in harmony with this stricter statement of his results. It is a warning "against any hasty conclusion that the neural mechanisms which synthesise reflex movements illustrate in their arrangement also those concerned where sensual fusion is the phenomenon" (p. 386), and a demonstration of "the different value and directness of spatial union of conducting-paths for motor synthesis and for psychical respectively" (p. 384). That is, we are warned against the common assumption that, as in the phylogenetic process, the physiological units (at the lowest level the cells, at a higher level the metameres) become physiologically and structurally compounded to form a more completely integrated organism, so the psychical life of the ascending series represents the increasing fusion of originally independent psychical lives of the physiological units; and we are prepared to entertain the opposite view, namely, that as, in both the ontogeny and the phylogeny, the evolution of the organism consists in a continued differentiation of organs and functions from the primitive unicellular organism in which all functions are combined in rudimentary degree and fashion, so the psychical ontogeny and phylogeny may consist, not in a progressive fusion of discrete mental atoms, but in a continued differentiation of a psychical life, which, from the first, was unitary and contained potentially all the capacities which have been realised step by step with the evolution of the nervous system.

The former view comports with the doctrine that consciousness is a mere epiphenomenon, the latter with the view, to which Sherrington inclines (p. 390), that psychical processes play a part of increasing importance throughout the ascending scale of life; and that, in some way which at present wholly eludes our comprehension, the psychical life, which is from the first integrated, presides over the progressive integration of the nervous system, which, keeping pace always with its increasing differentiation, enables it to perform its supreme function, the integration of the complex bodily organism.

W. McDougall.

*Traité des Torticolis Spasmodiques : spasmes, tics, rythmies du cou, torticolis mental, &c.* Par RENÉ CRUCHET. Pp. xv., 836. 120 illustrations. Paris : Masson, 1907. Price 15 francs.

No better proof is required of the progress of neurology than the appearance of this splendid monograph of 800 pages on spasmodic torticollis and the various tics and spasms that affect the muscles of the neck. For two reasons the volume is noteworthy. In it will be found reproduced *in extenso* practically all the cases of torticollis that have hitherto been recorded, amounting to no less a number than 357. The documentary value of these cases can scarcely be over-estimated, representing as they do much patient research and selective care, in addition to considerable linguistic ability ; for all, needless to say, have been translated into French. In the second place, as is remarked by Professor Pitres in the preface which he contributes, the book is a successful attempt to dispute the old idea that there is a nosographical specificity in spasmodic torticollis. The merest tyro in medicine can make a diagnosis of spasmodic torticollis, but a diagnosis which is arrested there is more insufficient than ever, in view of the information which this work supplies. It is generally admitted that the treatment of torticollis is frequently unsatisfactory to an extreme degree ; in the presence of those distressing cases of generalised spasmodic movements known as bradykinesis, there must be few who are not conscious of therapeutic helplessness. Our confession of ignorance and our indecision of therapeutic method would be rarer if our knowledge were based on a more adequate appreciation of what may be included in the one term " torticollis." The confusion that exists to-day on these points is notorious : tic douloureux and writer's cramp are conditions differing widely enough, yet if the morbid process underlying each were to engage the muscles of the neck, forms of torticollis would be produced which, to an uneducated eye, would appear identical. The debt which we owe to the French school of neurology for its alternately analytic and synthetic work in the elucidation of functional disease is as great to-day as in the time of Charcot. Out of the farrago of torticollis, Cruchet has endeavoured to extricate various conditions differing radically one from the other, but alike in that their incidence is on the neck musculature. From the therapeutic standpoint, therefore, his analysis is all-important.

By the term "spasmodic torticollis," Cruchet implies spasmodic or convulsive movements, tonic, clonic, or tonico-clonic, intermittent in character, and involving the muscles of the neck, and his sub-divisions are as follows: (1) neuralgic torticollis; (2) professional torticollis; (3) paralytic torticollis; (4) true spasmodic torticollis; (5) rhythmic torticollis; (6) tics of the neck; (7) habit torticollis and mental torticollis.

### (1) *Neuralgic Torticollis.*

Corresponding to the tics and spasms of the face that accompany facial neuralgia are the spasmodic movements, forms of torticollis, that accompany occipital neuralgia. The sudden onset of acute pain at some localised spot in the occipital region, radiating to shoulder or face or arm, is followed by the appearance of a spasmodic torticollis usually tonic in form, sometimes clonic. As soon as the neuralgic pain ceases, the head twitches die down, the stiffness of the neck disappears, and the patient regains control of his muscles.

Spasmodic phenomena occur in facial neuralgia in between one third and one half of the cases; and while occipital neuralgia is perhaps three or four times less frequently met with than the former, the number of cases of it that are accompanied by spasmodic reactions is proportionately the same. The motor phenomena of neuralgias in general are both voluntary and involuntary. Hence, in the first of these, we meet with varieties of torticollis which it is difficult not to believe are attitudes assumed voluntarily to arrest pain. On the other hand, we see cases of torticollis the motor part of which is strictly comparable to the involuntary movements of so-called tic douloureux. Of what nature are these involuntary movements? Cruchet maintains stoutly, contrary to the opinion of Meige, that tic douloureux is a true tic and not a spasm. The characters of a spasm, *i.e.*, of a movement produced by irritation of a motor nerve, are tonic and clonic twitches, sometimes synchronous, vermicular movements, fascicular contractions, which cannot be reproduced voluntarily, but can be by electrical stimuli; they habitually persist in sleep. Tic douloureux, however, is a "figured" movement, an act of defence, a movement which can be simulated voluntarily, and indeed arrested momentarily. It is affirmed, then, that neuralgic torticollis is frequently of the nature of a tic douloureux; and if the convulsive movements continue independently of the attacks of pain, we may find a tic non-douloureux supervening, a habit torticollis, to which reference is made in another chapter.

(2) *Professional Torticollis.*

This term is employed for those tonic, clonic, or tonico-clonic types of torticollis which occur only at the moment of execution of a given functional or professional act which concerns the muscles of the neck; at all other times the movements of these muscles are perfectly normal. Torticollis of this description has been rather lost sight of; yet it deserves special mention, for it exactly resembles writer's cramp, though it forms but a minority in the spasmodic torticollis syndrome. A variety of this type is constituted by torticollis which develops only when the patient is walking or standing, though cases which come under this category are probably very rare. The occupations in which torticollis of this nature is specially apt to appear are, according to our French author, those of cobbling, tailoring, and paving; in the majority of instances, however, the individuals affected are those who for pleasure, or by profession, read and write a great deal, and who eventually suffer from writer's cramp. The combination is by no means infrequent. From the pathogenic point of view, the processes are identical, and no adequate evidence has ever been offered to oppose the hypothesis of Duchenne that the trouble is central in origin. As with neuralgic torticollis, the movements may become independent of the professional act, in which case they persist as post-professional torticollis and may pass into the true spasmodic type.

(3) *Paralytic Torticollis.*

Paralytic torticollis is produced by the more or less widespread paralysis of the muscles of the neck, and this paralysis may be central or peripheral in origin. Such peripheral palsies are practically unrecognised, a fact which in itself is sufficiently surprising, seeing that there can be little difference between the face and the neck as far as exposure is concerned. Facial spasm consecutive to facial palsy is an accepted condition, but we have not learned to expect a similar sequence in the neck musculature. Following on a chill, or a traumatism, or an infective disease, comes a certain stiffness or pain in the neck, which the patient ignores, but after an interval of some days or weeks he notices that his head is tilted to one side; the more this deviation is accentuated the less able is he voluntarily to correct it, save for a moment or two; at this stage he usually is driven to various subterfuges to aid him. It is of fundamental importance to note that there is no contracture (primarily at least), since the vicious attitude can be overcome; nor is the torticollis a convulsive one.

Naturally, one finds that the torticollis is more pronounced when the patient is on his feet or walking, as the neck muscles then enter into bilateral action to support the cephalic extremity on the vertebral column. By over-functioning and hypertrophy of the non-paralysed sternomastoid the paralytic torticollis may develop into the true spasmodic variety.

(4) *True Spasmodic Torticollis.*

Here a subdivision is desirable into idiopathic spasmodic torticollis and symptomatic spasmodic torticollis.

With idiopathic spasmodic torticollis we all are familiar. It may be remarked that the contractions are variable in duration and intensity, that they may be tonic, clonic, or tonico-clonic, that we constantly find certain muscles rigid, and that a varying and inconstant degree of pain accompanies this rigidity. The muscular contractions themselves present the characters of true spasms as already detailed: *i.e.*, the whole of a muscle involved does not contract at once; the contractions are fascicular; associated movements are common; the platysma, the face, the muscles of the shoulder, are often implicated. The influence of the will on this condition is practically *nil;* antagonistic gestures are frequently inefficacious. Nevertheless, we may find that pressure on certain points arrests the spasm; the explanation of the action of this pressure is still to seek. It is quite exceptional to remark any mental defect in cases of true spasmodic torticollis. The pathogeny of this type, this hemispasm of the neck, is analogous to that of facial hemispasm. Of the numerous hypotheses which have been advanced, Cruchet declares for the view that the lesion is in the peripheral nerves, and that it is possibly neuritic.

In symptomatic torticollis the disease has been attributed to alcoholism and various intoxications, to influenza, rheumatism, typhoid, diphtheria, &c. Special mention ought to be made of malaria. Spasmodic torticollis may be one of the localised symptoms of an epileptic attack, the *epileptic trachelismus* of Marshall Hall. Or it may be a true epileptic equivalent. Spasmodic torticollis of cortical or subcortical origin is very debateable. Diffuse spasmodic torticollis—spasmodic bradykinesis—is supposed to be central or medullary in origin.

(5) *Rhythmic Torticollis.*

By this term are meant simple or combined movements which, in a given individual and for a certain time, recur at equal intervals

and are alike in rate and intensity. This question forms a large part of the subject, and it is handled by Cruchet admirably. Space will not allow detailed discussion of many points of interest that suggest themselves. The reader will find much of value on spasmus nutans, eclampsia nutans, functional and hysterical tremors, the rhythmical movements of idiocy and epilepsy, &c. The occurrence of rhythmical movements of the head in toxæmias, such as typhoid fever, meningitis, erysipelas, tetanus, alcoholism, paludism, is well recognised. The increase of these movements in proportion to the temperature of the patient is significant. Rickets and spasmus nutans are intimately connected. Reference must be made to a topic which Cruchet has made specially his own, viz., the rhythmical movements of children in sleep. All these are treated with a wealth of clinical illustration.

(6) *Tics of the Neck and Mental Torticollis.*

Little need be said in this place of tics of the neck; they ought to be readily distinguished from the conditions that have been enumerated. The standard work of Meige and Feindel has led to a clear understanding of their nature, and Cruchet merely amplifies and applies the knowledge which neurology has gained by the labours of the French school. It is impossible to do more than mention that he does not believe in the existence of the tonic tic, considering it a contradiction in terms, but in reality his disagreement with Meige is reduced to one of nomenclature. The chapter on habit torticollis and mental torticollis deserves reading *in extenso*. The former expression is used to indicate attitudes or deformities of torticollic type due to ocular or otic defects, among others, or adopted for other reasons and become involuntary. Mental torticollis is the phrase imagined by Brissaud to explain cases where there is a preponderant psychical factor in the disease. That such a factor is present in some instances there can be no doubt, but in all probability the category of these cases has been unjustifiably enlarged. As Féré says, a psychical theory has the immense advantage of dispensing with every effort in search after a physical cause, but it has the disadvantage of destroying all chances of finding it.

We cannot, in the limits of a brief review, enter on a criticism that would be adequate. There is material for discussion on almost every page. Cruchet's interpretation of some of the published cases is questionable, but for this paucity of detail is to a certain extent responsible. The relation of torticollis to trauma is not, perhaps, emphasised enough. Mention might have been

made of a condition which might be called "torticolis circulaire," analogous to "folie circulaire." The physiological association of the sternomastoids and the abdominal muscles, and their involvement in disease, ought to be alluded to. There is little reference to the modifying influence of surgical interference, or to the development of other forms of spasmodic movement secondary to operation. Surgical treatment is decried *un peu partout*; the astonishing statement is made (and in italics too) that there is on record only one case of permanent cure after section of the posterior divisions of the upper cervical nerves.

By way of minor criticism it may be noted that Handfield Jones is referred to everywhere in the volume as Jones Handfield; that various other English authors are incorrectly named; where English words occur they are frequently misprinted ("subliminial" for "subliminal," "exercice" for "exercise," "fonctional" for "functional," "medicin" for "medicine," &c.).

The book combines solid scientific worth with a charming and lucid style, and everywhere gives evidence of the culture of its author. The historical sections are not the least fascinating in a treatise which will remain a standard work for many years to come, and which will stimulate interest in the unfortunate individual of whom a seventeenth-century poet wrote:—

> Mon pauvre corps est raccourci,
> Et j'ai la tête sur l'oreille:
> Mais cela me sied à merveille,
> Et parmi les torticolis
> Je passe pour des plus jolis.

S. A. K. Wilson.

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## Publications Recently Received.

[*Notes on a book under this heading do not preclude a subsequent review.*]

*Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie.* X. Jahrgang. Bericht über das Jahr 1906. Pp. 1,350. Berlin: Karger, 1907.

This book should be on the shelves of every working neurologist. No such work can ever be complete, but the time saved by its possession is incalculable. The arrangement has the same excellent characteristics as in previous years.

*Collected Papers on Circulation and Respiration.* First series, chiefly containing Laboratory Researches. By Sir LAUDER BRUNTON. Pp. 696. London: 1907.

The first volume of the collected papers of Sir Lauder Brunton contains those concerned with circulation and respiration. A short autobiographical preface adds greatly to the interest and value of the volume.

*La Neuronophagie.* Par Dr. RENÉ SAND. Pp. 156. Brussels: Hayes, 1906.

A treatise on the destruction of nerve-cells by phagocytosis. It opens with an extensive list of references to those authors who have described this condition, arranged according to the date of publication and the circumstances under which the process occurred. This is followed by an account of the author's researches. There is one coloured plate.

*The Labyrinth of Animals.* By ALBERT A. GRAY, M.D., F.R.S.E. Vol. i., pp. 198. London: Churchill, 1907.

This volume will be of the greatest interest to those interested in the comparative structure of the internal ear. The author has succeeded in removing the bone so as to leave the membranous labyrinth intact. Each specimen was then photographed stereoscopically. By means of a hand stereoscope the photographs can be combined, and the labyrinth stands out in all its complexity as a solid body. The book contains thirty-one illustrations.

*The Major Symptoms of Hysteria.* Fifteen lectures given in the Medical School of Harvard University. By PIERRE JANET, M.D. Pp. 345. New York: Macmillan, 1907.

A fascinating book which should be read by all interested in the analysis of hysteria. Janet derives all its graver phenomena from somnambulism and fractured personalities. Each chapter treats of some leading group of symptoms and shows how it also depends on the narrowed field of attention. In a further edition it would be well to remove gallicisms such as "diapason" for tuning-fork, and the use of "accident" in both its English and French meaning.

*Hypnotism or Suggestion and Psychotherapy.* By Dr. AUGUST FOREL. Translated from the fifth German Edition by H. W. ARMIT. Pp. 370. London and New York: Rebman, 1906.

An English translation of the well-known work of Professor Forel. It is full of information concerning the phenomena of hypnotism and the personal experience of the author in the practice of suggestion. The weakest portion is that dealing with the psychology of the hypnotic state. The translation is fair.

*Treatment by Hypnotism and Suggestion.* By C. LLOYD TUCKEY, M.D. Fifth edition, revised and enlarged, with an introductory chapter by Sir FRANCIS R. CRUISE, M.D., K.S.G. Pp. 418. London: Baillière, Tindall & Cox, 1907.

This new edition of Dr. Tuckey's book is considerably enlarged and contains the work of most recent authorities. The first nine chapters are devoted to the history of modern psycho-therapeutics and to a somewhat diffuse consideration of the nature of hypnotism. Then follows an account of conditions successfully treated by the author and by other practitioners.

*Hypnotism and Suggestion.* By EDWIN ASH, M.B., B.S. Pp. 134. London: Jacobs, 1906.

A popular book which makes no pretence at scientific treatment of the subject. It gives practical rules for the induction of hypnosis and for the use of therapeutic suggestion.

*La Psychose Maniaque-Dépressive.* Par G. DENY et PAUL CAMUS. Pp. 96, with ten figures. Paris: J. B. Baillière et Fils, 1907.

An interesting little book, giving a full account of the course and symptoms of circular insanity and of the various conditions under which it arises. It forms one of a series called "Les Actualités Médicales."

*Les Thérapeutiques Récentes dans les Maladies Nerveuses.* Par M. LAUNOIS et A. POROT. Pp. 96. Paris: J. B. Baillière et Fils, 1907.

Another volume from the same series, dealing with lumbar puncture and intraspinal injections, the treatment of tics, mer

curial injections in syphilis of the nervous system, arsenic in chorea, gaseous injections for the treatment of neuralgia, and lastly some recent surgical procedures.

*Die Melancholie: eine klinische Studie.* Von Dr. GEORGES L. DREYFUS. Mit einem Vorwort von Hofrat Professor Dr. EMIL KRAEPELIN. Pp. 329. Jena: Gustav Fischer, 1907.

The author determined to follow the subsequent history of those patients of the Heidelberg Institute for Mental Disease diagnosed as cases of melancholia. The result is a work of unusual interest, leading to the conclusion that, however extended the course, ultimately melancholia will be found in almost every case to be a phase of a maniacal depression or circular insanity. The book contains a wealth of material, clearly set out, which cannot be summarised in a short notice.

*Les Auto-Mutilateurs.* Par Dr. CHARLES BLONDEL. Pp. 132. Paris: Jules Rousset, 1906.

A short medico-legal and psycho-pathological account of self-mutilation; voluntary castration, enucleation of the globe (œdipism), combustion, and the injuries self-inflicted in agitated melancholic states are described. These chapters are followed by a consideration of the principal types and processes of self-mutilation, with a final chapter on self-inflicted injuries among soldiers.

*Grundzüge der Psychiatrie.* Von Dr. FRANZ C. R. ESCHLE. Pp. 297. Berlin und Vienna: Urban und Schwarzenberg.

An interesting text-book for those who are already familiar with some of the tendencies of modern thought in mental medicine. Stress is laid more on the psychological genesis of mental states than on gross anatomical changes. It follows almost entirely the teaching of Professor O. Rosenbach (Berlin).

*A Text-book of Psychiatry.* By LEONARDO BIANCHI. Authorised translation from the Italian by JAMES M. MACDONALD. Pp. 904, with 106 illustrations. London: Baillière, Tindall & Cox, 1906.

This book is divided into three parts, devoted respectively to the anatomy, physiological pathology, and clinical study of insanity. It is full and clearly written, with excellent clinical histories.

*Insanity and Allied Neuroses, Practical and Clinical.* By GEORGE H. SAVAGE and EDWIN GOODALL. New and enlarged edition. Pp. 624. London and New York: Cassell & Co., 1907.

This well-known work has been revised and enlarged. It still contains many interesting clinical histories, told in the authors' own inimitable style, and is an excellent introduction for students to the clinical and practical side of mental medicine.

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**EDITOR.**